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Effects of Dietary Manipulation on Patients with Peripheral Vascular Disease

by

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A Thesis

Submitted to the Faculty of Graduate Studies

and Research in Partial Fulfillment of the

Requirements for the Degree of

Master of Science

in

Nutrition

Faculty of Home Economics

Edmonton, Alberta

Spring, 1982

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research, for acceptance, a thesis entitled EFFECTS OF DIETARY MANIPULATION ON PATIENTS WITH PERIPHERAL VASCULAR DISEASE submitted by Linda Janet Whyte in partial fulfillment of the requirements for the degree of Master of Science in Nutrition.

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ABSTRACT

The effects of dietary manipulation on 42 patients (33 males, 9 females) with peripheral vascular disease were studied for a 12-month period. Patients were randomly assigned to either the American Heart Association Hyperlipidemia Diet C (AHA group, n=23) or a modified Pritikin diet (PRIT group, n=27). Patients were given one week of intensive dietary instruction in small groups and were then assessed once a month. They were encouraged to exercise regularly and to decrease their consumption of alcohol, tobacco, and caffeine. The dietary intakes showed that patients in the AHA group consumed approximately 49% of their energy intake as carbohydrate, 20% as protein, and 31% as fat and that they consumed 201 mg cholesterol and 23 g dietary fiber daily. The PRIT group had an energy distribution of 64% carbohydrate, 22% protein, and 14% fat and consumed 108 mg cholesterol and 43 g dietary fiber daily.

Generally, all patients showed decreases in fasting blood glucose, triglycerides, total cholesterol and low density lipoprotein (LDL) cholesterol and slight increases in high density lipoprotein (HDL) cholesterol. The PRIT group tended to show greater improvement than the AHA group in these parameters. The PRIT group achieved a significant decrease in serum cholesterol ($p < 0.01$) and serum LDL cholesterol ($p < 0.05$). The decrease in total cholesterol accompanied by no significant change in HDL cholesterol indicates an increased transport of cholesterol as HDL in these patients. It is thought that this could be due, in part, to the low fat intake and high fiber intake of this group. There was a consistent negative correlation between dietary fiber and serum cholesterol levels

($p < 0.01$). The AHA group achieved a significant increase in serum HDL cholesterol ($p < 0.05$). Average weight loss was 4.1 kg for the AHA group and 6.0 kg for the PRIT group. There was a general improvement in the length of time the patients could walk on a treadmill before experiencing claudication. This improvement was significantly greater for the PRIT group than for the AHA group at six months. The researchers have concluded that both dietary regimens, combined with exercise, can be of benefit to PVD patients.

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LIST OF ABBREVIATIONS

AHA - American Heart Association

CHD - coronary heart disease

CHO - carbohydrate

CHOL - cholesterol

DF - dietary fiber

F - females

HCF - high carbohydrate fiber diet

HDL - high density lipoprotein

HLP - hyperlipoproteinemia

IDL - intermediate density lipoprotein

LDL - low density lipoprotein

M - males

PRIT - Pritikin diet

PRO - protein

P/S - polyunsaturated:saturated fatty acid ratio

PUFA - polyunsaturated fatty acid

PVD - peripheral vascular disease

RBW - relative body weight

SFA - saturated fatty acid

TG - triglyceride

TVP - textured vegetable protein

VLDL - very low density lipoprotein

INTRODUCTION

Peripheral vascular disease is one of many health problems to which diet may be a contributing factor. Diet affects the lipid components in the blood, which in turn may affect the development of atheromatous plaques in the arteries.

Various dietary components, among them energy, fatty acids, cholesterol, carbohydrate, dietary fiber, alcohol and caffeine, have been investigated to determine whether they are related to blood lipid levels and atherosclerosis. Most studies of diet and vascular disease have been done only for short periods. The present study was conducted to examine the effect of two diet and exercise regimens on blood lipids and blood flow in patients with peripheral vascular disease over a twelve-month period.

CHAPTER ONE

LITERATURE REVIEW

Effects of Dietary Manipulation on Patients with Peripheral Vascular Disease

Peripheral vascular disease (PVD) occurs when there is insufficient blood flow through the arteries to the limbs. In most cases, this results from atherosclerosis. Epidemiological and experimental work on atherosclerosis has primarily been concerned with the coronary arteries; peripheral vascular disease has been studied less extensively.

The occlusion of arteries by atherosclerosis results from the focal accumulation in the intimal lining of a variable combination of lipids, complex carbohydrates, blood and blood products, fibrous tissue and calcium deposits. Cholesterol is the predominant lipid component (Smith, 1965; Smith et al., 1967). Triglycerides are present only in small amounts.

Patients with proven peripheral vascular disease tend to have high plasma levels of very low density lipoproteins (VLDL) and low density lipoproteins (LDL) and low levels of high density lipoprotein (HDL) (Bradley et al., 1978). When the cholesterol content of human mesenteric arterial wall was directly measured by fluorimetry, there was a positive correlation with plasma LDL cholesterol and a negative correlation with HDL cholesterol (Bonyers et al., 1976). Nestel and Poisner (1978) found a relationship between the cholesterol content of the human atrium and plasma lipoprotein levels. They obtained arterial biopsy specimens at the time of coronary artery surgery.

Patients with tissue cholesterol values in the upper quartile had higher plasma VLDL and LDL concentrations and lower HDL concentrations than patients with less tissue cholesterol.

Lipids in the arterial wall are derived primarily from the circulation (Zilversmit, 1968). Development of atherosclerotic lesions depend, at least in part, upon the transport of lipids to and from the arterial tissue. Clinically, a correlation between hyperlipoproteinemia and atherosclerosis has been established: high levels of low density lipoproteins (LDL) have been associated with rapid development of atherosclerotic plaques (Scanu, 1979). Serum cholesterol is correlated with low density lipoprotein levels in arterial intima (Smith, 1972; Onitiri et al , 1976). Niehaus et al. (1977) studied 16 patients undergoing arterial surgery. By labelling lipoproteins they showed that LDL entered the intima from the plasma. They also found that the net flux of LDL into the intima increased with age. Up to age 50, LDL levels are a strong predictor of risk of atherosclerosis and an even stronger predictor from 50-80 years of age. Zilversmit (1973) has suggested that when serum very low density lipoproteins (VLDL) are elevated, cholesterol-enriched particles may enter the arterial wall. In general then, there is evidence that elevated plasma levels of LDL and VLDL contribute to the development of atherosclerosis. High density lipoproteins (HDL) have more recently been implicated as a factor inhibiting atherogenesis (Miller and Miller, 1975). Patients with relatively low serum HDL reportedly have increased susceptibility to atherosclerotic disease (Hsia et al., 1975; Gofman et al, 1966). Since HDL transports cholesterol from the peripheral tissues to the liver, low HDL levels may mean decreased

capacity for cholesterol removal. Low serum HDL levels have been found to be more prevalent in patients with primary hyperlipidemias, in those surviving myocardial infarctions, in patients with peripheral vascular disease and in male heavy smokers (Eriksson and Carlson, 1974). Before menopause, U.S. women have a heart attack rate only a fraction of that of men of the same age. Women of this age also have high HDL levels. It is believed that these two facts are related (Kannel et al, 1971). In dogs and rats, two species with high levels of HDL, atherosclerosis is rare (Eisenberg and Levy, 1975). In people with Tangier's disease, a genetic abnormality where no HDL are present, there is a high degree of atherosclerosis (Frederickson, 1967).

Carew and associates (1976) summarized evidence for the negative correlation between the plasma concentration of HDL and the risk of atherosclerosis. They found that when arterial smooth muscle cells are incubated with LDL there is a rapid uptake of cholesterol. High HDL concentrations in the lumen show a protective effect by inhibiting the uptake of LDL and therefore decreasing cholesterol accumulation in the arterial wall. HDL also initiates the transport of cholesterol from the cells to the liver for excretion. The enzyme lecithin: cholesterol acyltransferase esterifies cholesterol and thereby enables it to be removed from the cell to the plasma. HDL are required for this enzyme to perform its function. These two mechanisms account for the protective effect of HDL. HDL cholesterol is being transported away from the atherosclerotic lesion while virtually all other cholesterol in the blood is going toward the

lesions to further complicate the disease. Plasma HDL represents the most powerful single lipid indicator of risk of vascular disease, especially in those over 50 (Kannel et al., 1979).

Patients with peripheral vascular disease often have elevated serum lipid levels. Lewis (1974b) found similar frequencies of elevated serum lipids in patients with peripheral vascular disease and in those with ischemic heart disease. In one study he found the percentages of combined hyperlipidemia (hypercholesterolemia and hypertriglyceridemia together), hypercholesterolemia alone and hypertriglyceridemia alone in patients with peripheral vascular disease were respectively 11%, 15% and 16%. Various studies have been done on the frequency of hyperlipoproteinemias (HLP). This is usually an elevation of levels of one or more lipoproteins due to some metabolic defect. Five major hyperlipoproteinemias have been identified. These may be primary (genetically determined) or they may be secondary to other diseases or the result of dietary habits. The five hyperlipoproteinemia phenotypes were classified by Frederickson et al. (1967). Vogelberg and associates (1975) found that patients with P.V.D. display hyperlipoproteinemia in 25-74% of the cases. Slack (1969) studied familial hyperlipoproteinemias and found that in males, Types III, IV and V HLP were more often associated with peripheral vascular disease than Type II HLP.

By lowering plasma lipid levels it may be possible to achieve regression of atheromatous lesions. Jagannathan and co-workers (1974) studied cholesterol kinetics in severe human atheromatous

lesions and found a turnover time of one to two years. This suggests the need for long-term lipid-lowering regimens. Recent work indicates that atheromatous lesions can regress and that it may be possible to induce this regression of atherosclerosis by dietary treatment. Armstrong (1970), working with rhesus monkeys, showed that atherosclerotic lesions developed during 17 months of high cholesterol feeding and regressed after 40 months of cholesterol-free diet. Barndt et al. (1977) studied patients with mild femoral atherosclerosis. Medical treatment with diet and drugs was used to reduce blood lipid levels. The atherosclerosis regressed as the hyperlipidemia was corrected. Pritikin and McGrady (1979) claim that a dramatic regression of atherosclerosis can be produced by means of a diet very low in fat and cholesterol and very high in fiber and starch, along with regular exercise. In patients suffering from PVD, they found a significant improvement in maximum walking distance and angiographic evidence of reversal of atheromatous plaques (Pritikin et al., 1975).

Several dietary factors influence serum lipid levels; some patients are abnormally sensitive to one or more of these factors. Among the factors of importance are: total energy, fatty acids, cholesterol, carbohydrate, dietary fiber, alcohol and caffeine.

A. Energy Intake

Excess dietary calories are stored as fat, resulting in obesity. Obesity promotes a greater fatty acid turnover. More VLDL is secreted by the liver and thus triglyceride levels increase. Albrink and co-workers (1962) considered weight gain and serum triglycerides

in 215 healthy males between 30 and 69 years of age: the 78 men who had lost weight or gained less than 4.5 kg since age 25 had a mean serum triglyceride level of 4.6 meq/liter (134 mg/dl). The 137 men who had gained more than 4.5 kg since age 25 had a significantly higher mean triglyceride level of 7.1 meq/liter (207 mg/dl). The men were divided into groups according to whether they had a family history of coronary artery disease or diabetes. Those with such a history had higher triglyceride concentrations than those without, suggesting a genetic factor. Often, a caloric restriction that decreases weight is enough to lower triglyceride levels. Thompson et al. (1979), Gotto et al. (1978), Olefsky et al. (1974) and Galbraith et al., (1966) found significant decreases in triglyceride levels with weight loss. It is possible that hyperinsulinemia is the link between obesity and hypertriglyceridemia. Obesity is associated with insulin resistance, and hence with excess insulin production. Excess insulin sometimes increases the production of VLDL and consequently may increase IDL and LDL. Bierman (1972) states that a controversy exists over the association between hyperinsulinism and hypertriglyceridemia and that the effect cannot be generalized. In Thompson's study, there was no change in LDL levels after a 10-week weight loss program. HDL levels decreased slightly, but after eight months they had returned to normal even though weight loss was maintained.

The National Diet and Heart Study (American Heart Assn. Monograph #18) (1967) showed a tendency for hypercholesterolemia to be more common

with increased relative weight. The 10% of the patients with highest relative weights were grossly hypercholesterolemic about twice as frequently as the 10% of those with the lowest relative weights. In an epidemiological study of over 40,000 people, Van Houte and Kesteloot (1971) found that cholesterol levels increased up to weights of 100 kg, but they decreased at higher weights. Studies have revealed contradictory effects of weight loss on cholesterol levels. A group of HLP patients attending a lipid clinic for two years who showed a significant and sustained weight loss, had no significant reduction in cholesterol levels (Gotto et al., 1978). In another study by Thompson and co-workers (1979), 15 obese females lost an average of 8.6 kg over a 10-week period. Again, there was no significant change in cholesterol values. On the other hand, Olefsky and co-workers (1974) followed 36 subjects until each had lost approximately 11 kg (2-10 months). They showed decreased cholesterol values (21%), decreased triglyceride values (44%), a 33% drop in insulin resistance and a 40% decrease in VLDL-TG production rates. The diet was simply low in calories (600-1600/day) with a composition of 43% of calories from carbohydrate, 15% from protein and 42% from fat. Six obese patients followed a 900-calorie reducing diet for 6-8 weeks and lost 4-7 lb/week (Galbraith et al., 1966). During the first 3-4 week period, the 900 calories included 1400 mg of cholesterol, yet serum cholesterol decreased an average of 55 mg/100 ml. The 900-Calorie diet for the second 3-4 week period contained no cholesterol, yet the serum cholesterol levels showed no further change. Various

authors have suggested that obese individuals may synthesize more cholesterol but also excrete more, thereby maintaining a relatively stable plasma level.

Garn and co-workers (1979) studied the relationship between fatness and blood lipid levels. In reviewing many studies they found that sample sizes are often too small. Sex-specific samples in excess of 500 would be necessary for correlations of approximately 0.1 to 0.3. Age effects on fatness-lipid relationships must be taken into account. Garn et al. reviewed data of 4,000 adults from the Tecumseh Community Health Study, aged 20-55 years. They found systematic but low order correlations between measured fatness and cholesterol in adults (males +.21, females +.11) and somewhat higher correlations for triglycerides (males +.29, females +.28). Thus, the contradictory results are likely due to small sample size, lack of consideration of age and sex effects, and questionable measures of fatness. Also, genetic factors may play a role. It appears that there will always be considerable individual variation in small-scale lipid studies.

B. Dietary Fatty Acids

Saturated fatty acids raise serum cholesterol levels whereas polyunsaturated fatty acids lower serum cholesterol. Saturated fatty acids vary in their hypercholesterolemic effect with the C_{12} to C_{16} compounds, lauric, myristic, and palmitic acids having the greatest. Stearic acid has little influence on serum cholesterol; however, stearic and palmitic acids increase triglyceride levels (Grande et

al., 1970, 1972). Although polyunsaturated fatty acids lower serum cholesterol levels, the effect is less marked than that of saturated fatty acids in raising serum cholesterol. The mechanism of action of polyunsaturated fatty acids has not been elucidated. There is considerable support for the contention that polyunsaturated fatty acids increase bile acid excretion and perhaps neutral sterol excretion. Other possibilities include the reduction of cholesterol absorption in the gut, decreased endogenous cholesterol synthesis, diversion of circulating cholesterol into the tissues and reduction of circulating levels of low density lipoproteins.

C. Dietary Cholesterol

There is wide variation among individuals in response to cholesterol in the diet. Dietary cholesterol has a hypercholesterolemic effect independent of fatty acid composition of the diet. Patients fed as much as 3 grams cholesterol per day absorb up to one gram per day. The absolute absorption appears to be greater when intake is increased, although the fraction absorbed declines (Keys, 1965). Quintao and associates (1971) have shown that in many, but not all individuals the effect of prolonged high intake of cholesterol on exchangeable cholesterol pools in the body is minimized by two compensating mechanisms:

(1) enhanced fecal excretion of neutral sterols (Grundy and Ahrens, 1969)

(2) suppression of endogenous synthesis of cholesterol (Grundy et al., 1969).

Cholesterol entering the intestine seems to exert a negative feedback effect on cholesterol production in the liver. However, if less than 300 mg cholesterol is consumed each day, plasma cholesterol will fall considerably despite the increase in endogenous cholesterol synthesis (Mattson et al., 1972). Populations consuming little cholesterol show a very strong, almost linear correlation ($r = 0.87$) between dietary cholesterol and plasma concentrations.

Connor reported on lipid status in a population study of a group of Tarahumara Indians who had an average daily cholesterol intake of 71 mg (range:17-144 mg) and a mean plasma cholesterol level of 125 $\pm$ 26 mg/100 ml (Connor et al., 1978). LDL cholesterol levels averaged only 87 mg/100 ml, while HDL levels averaged 25 mg/100 ml. The latter value is not high enough to be considered protective, but presumably it is low because of the low total cholesterol value. The diet of the Tarahumaras was low in cholesterol (71 mg/day), low in fat (12% of total calories) and low in saturated fat (2% of total calories). Protein and carbohydrate comprised 13% and 75% respectively of the total calorie intake. The crude fiber intake averaged 19 g/day. Connor and Connor (1977) studied 25 subjects who had been previously consuming a "typical American diet", high in fat, high in cholesterol. Initially, when these subjects were fed a cholesterol-free diet for 3-4 weeks, mean plasma cholesterol levels decreased from 251 mg/dl to 211 mg/dl. Then they were fed a diet containing 1000 mg cholesterol per day for another three to four weeks. Cholesterol response to the dietary changes is shown in Table 1.1. Distinction is made between normal subjects and those with Type II and Type IV

TABLE 1.1 The plasma cholesterol levels (mg/dl) after the usual American diet and cholesterol-free and high-cholesterol diets in normal and hyperlipoproteinemic subjects.

	Period I			Period II		
	Usual American Diet	Chol-Free Diet	change	Chol-Free Diet	High-Chol Diet	change
Normal Subjects	171 ± 8[1]	141 ± 14	-30	141 ± 14	174 ± 20	+33
Type IIa-mild	258 ± 18	209 ± 49	-49	209 ± 27	245 ± 29	+36
Type IIa-severe	375 ± 22	338 ± 43	-38	338 ± 43	405 ± 17	+67
Type IV	251 ± 36	208 ± 23	-43	208 ± 23	231 ± 23	+23
Mean (all subjects)	251	211 ± 68	-40	211 ± 68	247 ± 79	+36

[1]Values are means ± S.D.

SOURCE: Connor & Connor, 1977

HLP. A mean decrease of 40 mg (16%) occurred on the cholesterol-free diet. When 1000 mg cholesterol per day was added, a mean increase of 36 mg (17%) occurred. The increased dietary cholesterol produced the greatest elevation of the LDL fraction in normal and Type II subjects. Those people with Type IV HLP had increases of VLDL and LDL (almost 50% from each).The authors suggested that the occurrence of a metabolic block in the conversion of VLDL to LDL was the possible cause.

Brown (1966) has studied the interaction between the effects of intake of polyunsaturated fatty acids and cholesterol on serum cholesterol concentration. The critical dietary levels for effective serum cholesterol reduction have been established. When cholesterol intake is low, less increase in polyunsaturated fatty acid intake is needed to achieve a satisfactory reduction in serum cholesterol.

Table 1.2 shows the results of studies conducted to lower serum cholesterol levels by altering the fat composition of the diet. Generally it appears that lowering total fat and cholesterol and increasing polyunsaturated fat in the diet decreases serum cholesterol and LDL levels.

D. Dietary Carbohydrate

Dietary carbohydrate in the form of simple sugars influences serum lipid levels, particularly triglyceride levels (Albrink, 1973; Antonis and Bersohn, 1961). In some lipidemic subjects a high sugar intake produces more intense lipemia with triglyceride levels increasing by 1000 mg/dl or more (Anderson, 1967). There is, however, considerable individual variation in lipid response to carbohydrate

Table 1.2 The effect of dietary fat and cholesterol on plasma lipids in human studies.

Reference		Diet	Subjects	Results
Lyon, T.P. et al.(1956)	a	50 g fat 200 mg chol.	155 M I[c] Patients	-15 re-infarcts decreased LDL[d]
	b	Regular diet (Reg)	125 M I Pts. 4 years treatment	-51 re-infarcts
Nelson, A.M. et al. (1965)	a	50-60 g fat P.U.F.A.[e]	88 M I Pts.	-10% chol (2/3pts) 35% CHD Death
	b	Reg. diet	754 M I Pts. 10-13 yrs. treatment	-79% CHD Death
Morrison, L.M. (1960)	a	20-25 g fat 50-70 mg chol. 225 g CHO 120 g PRO 1600 cal.	50 M I Pts.	-30% chol 8 yr.-44% mortality 12-yr.-6% mortality
	b	Reg. diet	50 M I Pts.	-8 yr.-76% mortality -12 yr.-100% mortality
Koranyi, A. (1963)	a	35-40 g fat	M I Pts.	-3 yr.-8.6% mortality
	b	Reg. diet	M I Pts.	-3 yr.-19.7% mortality
Hood, B. et al. (1965)	a	50-60 g veg oil fat chol	112 M I Pts.	-Chol-7% (M) -20%(F)
	b	Reg. diet	112 M I Pts.	-Chol-14%(M) -6% (F)

Table 1.2 CONTINUED

Reference	Diet	Subjects	Results
Leren, P. (1970)	a Fat Chol P.U.F.A. 264 mg Chol 39% fat 55 g P.U.F.A. 22 g SFA P/S ratio 2.4 b Reg. diet	30-64 yr.	-Cardiac events were significantly less ($p < 0.05$) in experimental group -chol[e] -15-20%
Stamler, J. (1971)	a 33% fat 400 mg Chol 1.6 g Na	941 M.I. pts.	-Chol of ≥ 260 new coronary events ↓ from 43%-→ 20%
	b Reg. diet	457 M.I. pts.	- ↓ from 38%-→ 32%
Wilson, W.S. et al. (1971)	AHA Diet	59 pts. (non-obese)	(1 mo.) Chol - 9.7% LDL - 12.6%[f] (6 mo.) Chol slightly > ↓ LDL - 17.3%
		8 Type II Pts.	(1 mo.) Chol no change LDL no change (6 mo.) Chol - 13.3% LDL - 20.5%
Bierenbaum, M.L. et al. (1973)	a 28% fat < 9% SFA < 400 mg Chol	100 (M) (30-50 yr.)	5 yr. ↓ Chol[d] 10 yr. ↓ Lipids[d]
	b Reg. diet	100 (M)	No Δ Chol
Anderson, J.T. et al. (1973)	a Moderate low Chol b Strict low Chol c High Chol		Chol / T.G. -17% / same -29% / +9% +9% / +25%
Porter, M.W. et al. (1977)	Reg. Diets + a limit of 1 egg/day b 0 eggs	114 (M) cross over design	No significant difference in Chol or T.G. N.B. only 200 mg. chol difference/day
Garcia-Palmieri et al. (1980)	a High fat high chol low CHO low starch b less fat and chol more CHO and starch	Urban 6,549 (M) (45-64 yr.) Rural 3,275 (M)	Higher Chol[d] than rural

a - experimental group
b - control group
c - M.I. = myocardial infarction
d - statistically significant
e - ↑ = increased ↓ = decreased Δ = change
f - greater response of LDL than cholesterol to AHA diet

feeding. Substituting simple sugars for starch may result in increased concentrations of serum triglycerides and serum cholesterol. It was thought that carbohydrate influenced triglyceride formation only in the liver, but den Besten and co-workers (1973) showed that the intestine may be an important site of regulation. Hall et al. (1972) concluded that a low carbohydrate diet is seldom required to achieve significant lowering of serum TG in middle-aged men, provided weight loss is accomplished and there is a sustained low intake of saturated fat and cholesterol. However, in some cases, a low fat, high carbohydrate diet results in hypertriglyceridemia (Ginsberg et al , 1976).

E. Dietary Fiber

Fiber components in the diet have been investigated for their effects on plasma lipids and steroid excretion. Generally, it is thought that the addition of fiber to the diet lowers lipid levels, but that this effect differs considerably with different types of fiber (Spiller and Kay, 1979).

Dietary fiber has many components. It is only recently that foods have been analyzed for dietary fiber and its constituents and therefore little is known about this subject. Most of the food composition tables provide figures for the crude fiber content of foods. Crude fiber is determined by a standardized method involving separation of insoluble material after treatment with hot acid and hot alkali. Crude fiber is only a part of dietary fiber, which is that component of food resistant to hydrolysis by the digestive

enzymes of man. A majority of this material is polysaccharides and lignin which are the structural components of plant cell walls (Fig. 1.1) (Yost, 1972; Aspinall, 1973). Lignin is deposited in the cell wall as the plant matures. It shows greater resistance to enzymatic digestion than other constituents of fiber.

Two methods have been used to determine dietary fiber:

(1) The method of Van Soest

(2) The method of Southgate

The method developed by Van Soest and McQueen (1973) measures only insoluble fiber components (cellulose, hemicellulose, lignin). The method developed by Southgate (Southgate, 1976; Southgate, 1969; Southgate et al., 1976) measures all fiber components, including water-soluble ones (pectins, gums, soluble and insoluble hemicelluloses, cellulose and lignin). Table 1.3 describes the characteristics of the constituents of fiber. The dietary fiber content of foods can be found in McCance and Widdowson's Food Composition tables (1978).

Dietary fiber is susceptible to some digestion by bacterial enzymes. The extent of this breakdown depends on:

(1) the nature of the bacterial flora of the colon.

(2) transit time

(3) physical composition of fibers.

(4) chemical composition of fibers.

Generally, vegetable cell walls are more fermentable than cereal brans since the latter have thicker cell walls that are more highly lignified. Wheat bran is one of the least digestible components of

TABLE 1.3 Chemical classification of dietary fiber

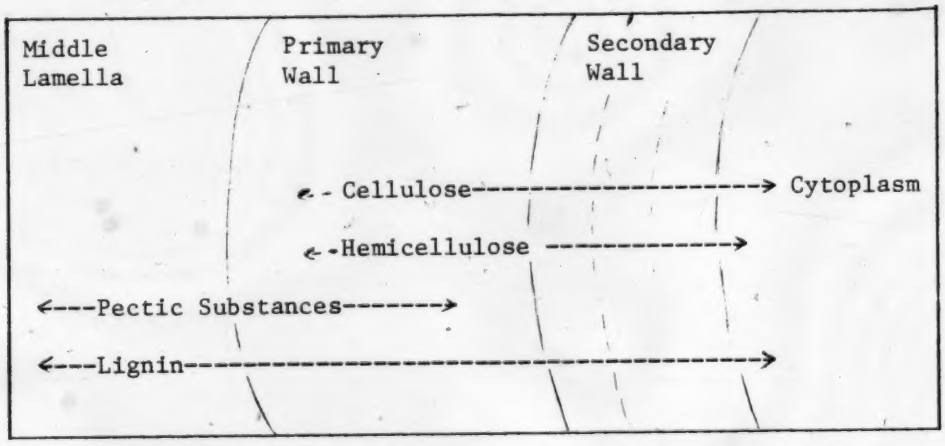
Fiber	Description	Major Chemical Components	
		Main Chains	Side Chains
Polysaccharides - Cellulose	Linear homoglycan[1] main structure component in plant cell wall: insoluble in concentrated alkali, soluble in concentrated sulfuric acid.	Glucose (1-4)	None
- Hemicellulose	Diverse group of heteroglycans[2] occurring in cell wall; vary in degree of branching and uronic acid content: soluble in dilute alkali.	Xylose Mannose Galactose Glucose	Arabinose Galactose Glucuronic Acid
- Pectic substances	Occur in primary cell wall and middle lamella (intercellular cementing substance); vary in degree of methylation.	Galacturonic Acid	Rhamnose Arabinose Xylose Fucose
- Gums	Formed by secretory cells and released at a site of injury to plants.	Galactose Glucuronic acid-mannose Galacturonic acid-rhamnose	Xylose Fucose Galactose
- Mucilages	Synthesized by plant secretory cells; occur in endosperm of plant seeds where they act to prevent excessive dissication: vary in uronic acid content.	Galactose-mannose Glucose-mannose Arabinose-xylose Galacturonic acid-rhamnose	Galactose
- Algal polysaccharides	Derived from algae and seaweed which differ in that cellulose in cellwall may be replaced by xylose and mannose; may be linear or branched-some types contain sulfated residues.	Mannose Xylose Guluronic acid Glucose	Galactose
Lignin	Non-carbohydrate: complex crosslinked phenyl propane polymer; 3 broad classifications corresponding to predominance of sinapyl, coniferyl, or coumaryl alcohols: probably has three-dimensional structure: infiltrates mature cell wall: resists bacterial degradation: insoluble in concentrated sulfuric acid.	Sinapyl alcohol Coniferyl alcohol Coumaryl alcohol	

[1] Containing one type of sugar residue.

[2] Containing more than one type of sugar residue.

SOURCE: Kay & Strasberg, 1978

Fig. 1.1 Structural components of plant cell wall[1]



[1] Middle lamella is primordial semifluid layer, high in protopectin content: primary wall is single layer, with 5-15% cellulose; secondary wall is multilayered with up to 50% cellulose.

Source: Kay & Strasberg, 1978, p.9

the diet: less than 30% is fermented in the colon, in contrast to the fiber found in peas (53%), corn germ (60%), sugar beet (65%), cabbage (66%) and carrots (74%) (Van Soest and Robertson, 1977).

Certain components of dietary fiber (lignin and pectin) have been shown to lower cholesterol. There are conflicting reports, however, of the effect of other components of fiber on blood lipids. Experimental work (1932-1976) on the influence of fiber intake on man was summarized by Kelsay (1978). Some forms of dietary fiber consumed in large quantity reduce serum cholesterol levels by 4% to 12%. The most effective sources are fruits and vegetables. Cholesterol levels were lowered by pectin (dosage: 6 to 36 g/day for 2-4 weeks); rolled oats (140 g/day for 3 weeks); guar gum (36 g/day for 2 weeks); legumes (100 g dry legumes/day for 3 months); beans (a sucrose replacement for 3 weeks) and a mixed diet of fruits, vegetables and legumes (19g crude fiber/day for 6 weeks). In only one study did wheat bran (20 g/day for 6 weeks) lower cholesterol levels, whereas ten studies showed no change with the addition of bran. Cellulose, whole maize, wheat and vegetables showed no cholesterol lowering. Wheat bran has, however, been found to have some effect on serum triglyceride levels. Serum triglycerides decreased when 18 to 100 grams of unprocessed bran was added to the diet (Kelsay, 1978). Changes in triglycerides generally did not occur when wheat fiber, bagasse, cellulose or pectin were administered.

Recently, Vahouny et al. (1980) noted that the issue is a complex one because of the many possible mechanisms of action of dietary fiber constituents on the absorption and metabolism of

cholesterol and triglycerides. They classified various types of fiber in terms of the following effects in rats (Table 1.4):

- decreased lymphatic absorption of cholesterol and triglycerides
- increased intestinal transit time
- binding of bile acids

The animals on diets containing alfalfa, cellulose and cholestyramine had significantly lower hepatic triglycerides and higher hepatic phospholipids. This may be a reflection of altered lipoprotein metabolism or turnover. The authors suggested that cellulose and wheat bran affect the bulk phase diffusion of lipids and interfere with their absorption. Thus cellulose and wheat bran may have an indirect effect on hepatic lipid and lipoprotein metabolism. Alfalfa meal contains pectin and lignin, components which bind bile acids in the intestinal lumen and increase the excretion of these metabolites as fecal acidic sterols. It was also observed that many of the fiber components affect triglyceride absorption to the same extent as cholesterol absorption.

Chen and Anderson (1979) found that feeding pectin, guar gum and oat bran to rats resulted in lower levels of serum triglyceride and cholesterol and in higher levels of high density lipoprotein cholesterol. They suggested that the depletion of bile acids by fecal excretion diverts cholesterol into bile acid pools and thus less cholesterol is available for incorporation into lipoproteins for release into the venous circulation (Anderson and Chen, 1979). It is thought that fiber may influence the size of chylomicrons,

TABLE 1.4 Effects of various types of fiber on cholesterol metabolism in rats

Type of Fiber (15% of diet)	Lymphatic Absorption of Triglycerides and Cholesterol	Transit Time	Bile Acid Binding
Wheat bran	Marginal decrease(↓)	Significant(↓)	-
Cellulose	Significant(↓)	Significant(↓)	-
Alfalfa Meal	Significant(↓)	-	Significant(↑)
Pectin	Significant(↓)	-	-
2% Cholestyramine (drug)	Significant(↓)	-	Significant(↑)
Yeast cell wall glycan	No effect	-	-

Source: Vahouny et al., 1980

thereby altering the proportion of cholesterol incorporated into the chylomicrons and VLDL and HDL.

Recent work done to study the effects of fiber intake on serum lipids and fecal steroid excretion is summarized in Table 1.5. It was found that cholesterol levels were lowered from 10 to 25%. These studies reported on a number of different sources of fiber, given to human subjects in variable amounts. There were large variations in response, as shown by the cholesterol values. Triglycerides were lowered by the HCF diet (see Table 1.5), .5g/kg wheat bran and 26g/day of soft or hard wheat bran, corn bran, soybean hulls or textured vegetable protein (TVP). Few studies considered lipoprotein values, but in one study it was found that VLDL and LDL were lowered and HDL was raised with an intake of 50g all bran cereal per day. In another study, LDL values decreased 21% with the addition of 26g hard wheat bran to the diet.

F. Alcohol Intake

Alcohol induced hyperlipidemia is characterized chiefly by hypertriglyceridemia. A large but transient increase in triglycerides can occur after ingestion of 8-10 oz of alcohol, and it has also been stated that moderate alcohol intakes may increase HDL. Castelli and co-workers (1977) examined data on alcohol and blood lipids of five major population groups. Conversion factors of Yano et al. (1977) were used to compute average amounts of absolute alcohol per week. They found a positive association between alcohol consumption and HDL cholesterol level in all of the study populations. There was a graded response even in the lower range of alcohol intake. Considering

TABLE 1.5. The effect of dietary fiber on plasma lipids in human studies

Reference	Diet	Subjects		Results	
Anderson, J.W. and Ward, K. (1977)	HCF - 40 g D.F. (natural sources-beans, oatmeal)			Chol - 50 mg	TG ↓
Kay, R.M. and Truswell, A.S. (1977)	controlled-pectin 15 g/day	4M 5F - 3 weeks		Chol - 13% + 44% fat excretion + 17% neutral sterol excretion + 33% fecal bile acids	TG no Δ
Kay, R.M. and Truswell, A.S. (1977)	high fiber-wheat bran 5 g/day crude fiber	3M 3F - 3 weeks		Chol no Δ - ↑ fecal bulk - ↑ fecal steroids	TG no Δ
McDougall, and Walker, K. (1978)	regular diet and 50 g all bran/day	9 pts. - 1-12 mo. 9 controls		Mo. 1 - no Δ in lipids Mo. 6-12 HDL Chol VLDL&LDL + 10 mg. no Δ - 29 mg.	
Anderson, J.W. and Chen, W.J.L. (1979) (review)				Lipids - % of Control Chol	TG
	17 g/day wheat bran	14		101	97
	25 g/day wheat bran	1		75	-
	12 g/day whole oats	1		89	-
	10.5 g/day bagasse	2		100	99
	16 g/day cellulose	3		100	-
	100 g/day cellulose	1		75	-
	9.6 g/day psyllium	2		87	-
	25 g/day pectin	10		87	99
	24 g/day guar gum	3		84	-
	bengal gram	1		78	-
van Berge - Henegouven, G.P. et al. (1979)	wheat bran .5 g/kg (33 - 38 g/day)	7M	4 wk.	Chol - 10%	TG - 24%
Munoz, J.M. et al. (1979)	16% Pro, 40% Fat, 44% Cho, 3 g crude fiber } 26 g soft wheat bran, 26 g corn bran	10M	28-30 days	Chol -	TG ↓
	+26 g soybean hulls			-	↓
	26 g TVP			- 14%	↓
	26 g hard wheat bran			-	↓
				- 12%	↓
Stasse-Wolthuis et al. (1980)				Chol	HDL
	low fiber - 18 g/day	40M	2½ wks.	-	-
	fruit & veg. - 43 g/day	22F	3 wks.	- 7 mg	no Δ
	citrus pectin - 28 g/day			- 13 mg	no Δ
	wheat bran - 37 g/day			+ 13 mg	no Δ
Anderson, J.W. et al. (1980)	HCF[1] diet (grains 40%) 64 g fiber/day (fruits 9%) (veg. 51%)	14M (lean)	days	Chol - 32%	TG - 11%
	HCF diet	11F	9-14 days	- 22%	no Δ
	HCLF[2] diet 23 g/day	9 (lean) 2 (obese)	9-12 days	- 20%	+ 28%

[1] HCF = high carbohydrate fiber diet

[2] HCLF = high carbohydrate low fiber diet

ll of the populations, a man who drank 5-6 oz alcohol per week ("moderate drinker")had an HDL level 10% higher than a man who did not drink at all. Triglycerides were positively correlated with alcohol consumption and there was a moderate to strong negative association between alcohol and LDL. The type of alcohol made little difference. Berg and Johannson (1973) studied the effects of ethanol on plasma lipid levels of healthy young medical students. They compared the effects of no alcohol intake to 63 ml per day of absolute alcohol as light beer (16% of total calories). Triglyceride levels increased in beer-drinking subjects after the first week of alcohol consumption. There was little change in plasma cholesterol and LDL levels. HDL significantly increased during the study and decreased after withdrawal of alcohol.

Glueck et al. (1980) did isocaloric substitutions of alcohol for carbohydrate in seven healthy male subjects. They found no significant changes in triglyceride or HDL. The alcohol injection period was for two weeks, which perhaps was too short a time span for obvious changes to occur. Nestel et al. (1976) showed that individuals varied in their susceptibility to alcohol-related hypertriglyceridemia and in some individuals the response to alcohol and carbohydrate was similar. In other individuals the response to alcohol was much greater than the response to carbohydrate or to carbohydrate plus fat. In these cases triglyceride levels decreased when alcohol was withdrawn. It is believed that the pathogenesis of hyperlipemia induced by ethanol intake may be due to an increase in liver triglyceride and apolipoprotein (Baraona and

Lieber, 1970). Alcohol has also been shown to decrease the catabolism of cholesterol to bile acids (Lefevre et al., 1972). Some underlying defect may be present in those subjects who are susceptible to alcohol-related hyperlipidemia.

G. Caffeine

Consumption of coffee has been linked to atherosclerosis. There is an elevation in plasma free fatty acids after caffeine ingestion (Graham, 1978). This is transient in nature, however, and little evidence of changes in lipids and lipoproteins has been found. Callahan et al. (1979) reviewed seven human studies which showed no consistent change in plasma lipids with excess caffeine intake. Nine similar studies in animals also proved to be negative. Reports of two international workshops on caffeine did not suggest any relationship of caffeine intake to either blood lipids or heart disease (Anonymous, 1979; 1980 a).

Much remains to be learned about the effect of various dietary components on blood lipid levels. There has been considerable argument as to whether atherosclerosis is solely due to lipid changes and lipids in the diet or whether it is related to a combination of dietary components. Several other factors are also thought to be involved in the etiology of vascular disease. These include:

- stress
- high blood pressure
- diabetes
- lack of exercise
- smoking

Lack of exercise and smoking are factors which may be modified to minimize their influence as risk factors (Gordon, 1977).

H. Inactivity

For many years, exercise has been recommended as a component of therapy for patients with vascular disease and it is believed to play a role in lowering blood lipids. Exercise also improves cardiac function, lowers blood pressure, increases fibrinolytic activity, retards platelet aggregation and results in weight loss if caloric intake remains constant. In populations which are physically fit, there appears to be some protection against the consequences of atherosclerosis. Of 600 people (350 of them being men over 40 years of age) from the Masai tribe, who were examined by Mann and co-workers (1972), only one man showed evidence of heart disease. Their serum cholesterol levels were low, rarely exceeding 150 mg/dl. Of 50 autopsies done on Masai men, not one death was due to heart disease. Paffenberger and colleagues (1970) studied 3,263 longshoremen over a 16 year period to determine the risk factors associated with deaths from heart disease. Cargo handlers who expended about 1000 kcal/day more than other longshoremen had a CHD death rate 25% lower than their sedentary work companions. This was true even when smoking, high

blood pressure, and overweight were present. The authors concluded that exercise may not prevent deposition of plaques, but it can cause the involved vessels to enlarge such that the relative degree of obstruction is decreased.

Exercises that require endurance are more beneficial in improving the cardiovascular system than exercises that produce increased muscle bulk. Exercise elicits its greatest response on blood lipids when the activity is strenuous and consistent. Lopez et al. (1974) studied 13 healthy medical students who participated in intense physical exercise for 30 minutes, four times a day. After seven weeks, serum triglycerides decreased an average of 27%, VLDL decreased an average of 42% and HDL increased an average of 16%. Cholesterol showed a moderate decrease. Holloszy and co-workers (1974) considered the effects of endurance exercise on the serum lipids of middle-aged men. Fifteen men between the ages of 35 and 55 (mean age 41.7 years) completed endurance calisthenics and distance running (two to four miles) an average of 3.35 times per week for six months. There was no significant drop in serum cholesterol. Triglycerides decreased an average of 40% but often, lipid-lowering is transient in nature and values increase when training ceases. This suggests the need for habitual exercise.

Oscai and co-workers (1972) have suggested a mechanism for the reduction of triglycerides through exercise. They state that exercise decreases the concentration of blood sugar. Carbohydrate therefore is diverted from the liver to the working muscles, resulting in a decreased availability of substrate necessary for lipogenesis.

Thus triglycerides and VLDL decrease. This occurs with acute exercise. With exercise of longer duration and greater intensity, glycogenolysis occurs and over the next several days carbohydrate is still being diverted to replenish glycogen stores. Thus TG and VLDL remain low. It is also thought that there may be further hydrolysis of TG and chylomicrons with exercise because the activity of the enzyme lipoprotein lipase, which catalyzes this reaction, also increases.

Often the hypocholesterolemic effect of exercise has been attributed to a caloric imbalance; however, many studies have shown decreases in cholesterol with exercise when there was no calorie deficit or weight loss. Generally it has been found that the LDL and VLDL fractions decrease and the HDL fraction increases with exercise (Hartung & Squires, 1980). Lopez and co-workers (1974) suggested that perhaps the increase in HDL overrides the decrease in LDL and VLDL resulting in minimal changes in actual total cholesterol values.

Two studies compared the effects of exercise alone, or exercise combined with dietary restriction, on vascular complications. Watt et al. (1976) followed postmyocardial infarction patients for 12 weeks. Group I (30 subjects) exercised three times a week for a total of 36 sessions. Each session involved a warm-up, walking, calisthenics and aerobics for a total of 45 minutes. Group II (30 subjects) participated in the same exercise regimen but they were also given a diet suited to their individual needs (fat controlled, energy controlled, Type IIB or Type IV diets). There were no significant changes in body weight. Group I (exercise alone) showed

no change in blood lipids. Group II (diet and exercise) showed mean decreases of 10% in cholesterol levels and 32% in triglyceride levels. Lampman et al. (1977) divided their sample of Type IV HLP patients into three groups - A (exercise alone), B (Type IV HLP diet) and C (diet and exercise). The Type IV diet was comprised of 20% protein, 40% fat (P/S=1.0) and 40% carbohydrate (30% sugars, 70% starches) at a caloric level for weight maintenance. The exercise component involved walking, jogging or cycling at 70% maximum heart rate for 30 minutes, three times a week. In all groups there was a reduction in blood lipids after six weeks. Triglycerides decreased 17%, 37% and 41% respectively in groups A, B and C. Cholesterol levels decreased 3%, 8% and 10% respectively in groups A, B and C. Diet and exercise appeared most beneficial in lowering blood lipids compared to diet alone or exercise alone. Although weight loss was minimal, significant decreases in body fatness occurred in groups A and C.

In patients with severe peripheral vascular disease, often the only possible form of exercise is walking. Improvements are measured by the length of time a patient can walk until pain occurs in the leg (claudication). Various factors contribute to the improvements in walking time. A training effect occurs which improves muscular strength and endurance in the legs. Also, it has been shown that after long-term exercise programs, collateral blood vessels develop in the legs which relieve or delay the onset of intermittent claudication. Concurrent dietary intervention can result in reductions in body fat and blood lipids, both factors which may improve circulation and possibly increase walking time. Pritikin et al. (1975) reviewed

the results of various walking therapies for PVD patients (Table 1.6). Again, the best results occurred when diet and exercise were combined. All studies showed improvement after a walking program was implemented. Most of them, however, did not specify changes in blood lipids. The Pritikin program of diet and exercise resulted in significant decreases in triglycerides and cholesterol.

I. Smoking

According to McIntosh et al. (1978), the evidence for a direct causal relationship between smoking and atherogenesis is far from secure, although it is believed that smoking increases the complications of atherosclerosis. The three main pharmacologically active agents in cigarettes are tars, nicotine and carbon monoxide. Because tar is not carried in the blood, it is believed to have little involvement in vascular disease. Nicotine can cause constriction of the blood vessels and thereby hampers the circulation. Carbon monoxide decreases the oxygen-carrying capacity of the blood.

Aronow and Isbell (1973) studied the carbon monoxide effect on a group of angina patients. On initial testing, the average exercise time on the treadmill was 226 seconds before angina pain was experienced. Then for two hours, the group was exposed to air composed of 50 ppm carbon monoxide. The average exercise time on the treadmill was then 187 seconds. In just two hours, their exercise tolerance decreased by 17%. Cigarettes produce more carbon monoxide than cigars and pipes and thus may have a more harmful effect.

TABLE 1.6 The effect of exercise on the severity of peripheral vascular disease in human studies

Reference	Number of Subjects	Age (Mean) Yrs.	Length of study (months)	Walking Distance (% gain from beginning)
Larsen, O.A. and Larsen, N.A. (1966)	14	57	6	280%
	(placebo 2x/day or walking daily until pain was experienced)			
Johannson, B.W. and Sievers, J. (1967)	10	62	6	300%
Zetterquist, S. (1970)	9	58	3-4	73%
	(daily walking - 1 hour)			
Pritikin U.S.A., 1975	15	60	6	302%
	(daily walking- 30-45 mins.)			
Pritikin U.S.A., 1975	18	61	6	5870%
	(daily walking - 30-45 mins.) nigh fiber, low fat diet			

Considerable epidemiological evidence has suggested a relationship between smoking and high blood lipid levels. In a male population study in Finland, Karvonen et al. (1959) found that the smokers had a higher average serum cholesterol than non-smokers. Experimentally, Pozner and Billimoria (1970) investigated this relationship. Sixty-four healthy volunteers, aged 19-30 years, were divided into non-smokers, light smokers and heavy smokers. Fasting blood samples were taken after a half hour rest period. Table 1.7 shows triglyceride and cholesterol values. The heavy smokers had significantly higher triglyceride (+28%) and cholesterol (+13%) values than those of the non-smokers. VLDL and LDL values were also higher. The effect of heavy smoking on raising serum cholesterol and LDL in females was much greater than in males. But the effect on raising VLDL was much greater in males than in females. It has also been stated that cigarette smoking lowers HDL levels (Anonymous, 1980,b).

J. The Role of Diet Therapy in Atherosclerosis

Dietary manipulation has been successful in lowering elevated serum lipid and lipoprotein levels. Many dietary trials have shown that serum cholesterol concentrations can be effectively lowered by 8 to 18% with diet (Leren, 1966; Turpeinen et al., 1968; Dayton et al., 1968; Wilson et al., 1971) in four to six weeks. A fat-modified diet, widely employed in the treatment of hyperlipidemia, is a regime that reduces saturated fat intake, partially replacing it with polyunsaturated fat. Cholesterol intake is also reduced.

TABLE 1.7 The effect of smoking on plasma lipids

	Group I (non-smokers)[a]	Group II (light-smokers)[b]	Group III (heavy-smokers)[c]
	n = 20	n = 17	n = 27
Sex	12M, 8F	6M, 11F	19M, 8F
Age	22.3	23.1	23.4
Triglycerides (mg/dl) (serum)	68.6 ± 6.0[d]	68.4 ± 5.4	87.6 ± 9.4[e]
Cholesterol (mg/dl) (serum)	176.3 ± 8.2	172.1 ± 6.7	200.0 ± 8.4[e]

[a] 0 cigarettes/day [b] $\leq$ 15 cigarettes/day [c] $>$ 15 cigarettes/day

[d] values are means $\pm$ S.E.

[e] significantly higher than non-smokers

SOURCE: Pozner and Billimoria, 1970.

Variants of composition have been designed with total fat intake ranging from 31 to 40% and a ratio of polyunsaturated to saturated fatty acids of 0.7 to 2.5. One widely used diet comprises protein 16%,, fat 37% (P/S ratio=1.8), carbohydrate 47% of total energy (Brown, 1966). Cholesterol intake is less than 300 mg/day. The diet is prescribed isocalorically for initially lean patients, or following a period of caloric restriction where weight reduction is indicated.

The transport of lipids in the plasma and lymph occurs in complexes stabilized by specific proteins as well as by more polar lipids such as phospholipids. The transport systems may be divided into:

- the albumin-free fatty acid complex
- high-density lipoproteins (HDL)
- low-density lipoproteins (LDL)
- very low density lipoproteins (VLDL)
- chylomicrons

The composition of these transport systems is shown in Table 1.8.

LDL cholesterol levels show an even greater responsiveness to dietary changes than total cholesterol. LDL transports 60% to 70% of circulating cholesterol and is strongly implicated in the pathogenesis of atherosclerosis on epidemiological and experimental grounds. The reduction in serum cholesterol achieved with the fat modified diet is due largely to lower levels of LDL cholesterol and to a lesser extent decreased VLDL cholesterol. The reduction in VLDL cholesterol consequently results in a fall in serum triglyceride levels.

TABLE 1.8 Composition of human serum lipoproteins[a]

Lipoprotein Class	Protein	Phospho-lipids	Cholesterol unesterfied	Cholesterol esterified	Triglyceri-ides
	Weight (percent per particle)				
Chylomicrons	2.0	7.0	2.0	5.0	84.0
VLDL	8.0	18.0	7.0	12.0	50.0
LDL	21.0	22.0	8.0	37.0	11.0
HDL_2	41.0	30.0	5.4	16.0	4.5
HDL_3	55.0	23.0	2.9	12.0	4.1

[a] Source: adapted from Shen et al., (1977). The data do not include the small amounts of glycospholingo-lipids reported by Dawson et al., (1976).

When hypertriglyceridemic patients are placed on fat modified diets, HDL levels usually rise (Carlson, 1977). Triglyceride levels have been found to be inversely related to HDL levels. In an investigation by Hulley et al. (1977), plasma HDL levels increased with a decrease in triglycerides, a reduction in smoking and a loss of body weight. Although cross-sectional studies have also shown plasma triglyceride values are inversely related to HDL in normal and hyperlipidemic populations, not all types of HLP respond in this way with treatment (Falko et al., 1979; Manninen et al., 1979). Spritz (1980) suggested that HDL levels increase with weight reduction.

Evidence is increasing which shows fat modified diets cause a decrease in serum cholesterol levels but these also cause a decrease in serum HDL levels (Shepherd et al., 1978; Lewis et al., 1981; Schaefer et al., 1981 and Vessby et al., 1980). It has been found that when high fiber intakes are added to fat modified diets, there is a selective lowering of serum total cholesterol concentrations while increasing serum HDL concentrations in both rats (Chen and Anderson, 1979) and humans (Lewis et al., 1981).

Many different diets have been designed to normalize serum lipid levels and lipoprotein patterns. Recent studies appear to have chosen more strict dietary treatment for hyperlipidemia than in the past, with emphasis placed on high complex carbohydrate, high fiber, and low fat intakes. High-fiber, low fat diets have been shown to produce dramatic reduction in serum lipid levels (Pritikin and McGrady, 1979). It is speculated that the increased fiber and

decreased fat components are both involved in the lipid-lowering effect. The extent to which each factor plays a role is uncertain.

Raymond et al. (1977) considered the effect of fiber and cholesterol by testing two groups of six subjects for two four-week periods on either a high fiber or fiber-free diet. One group also consumed 1000 mg cholesterol daily, while the other group consumed no cholesterol. Results showed that the high level of fiber intake led to no significant change in plasma cholesterol and triglyceride levels in either group. Those on the cholesterol-free diet had considerably lower plasma cholesterol levels than those on the high cholesterol diet (Table 1.9). In the latter, cholesterol elevations occurred primarily in the LDL fraction with a slight increase in HDL cholesterol also.

Significant improvements in blood lipids occurred in patients with peripheral vascular disease who, for a six month period, consumed a diet of fiber-rich unrefined starchy food (Pritikin et al., 1975). Total carbohydrate comprised 80% of the calories; fat was reduced to 10%; and protein was also at 10% of the total calories. The diet was low in cholesterol with no sugar or salt added to foods. In a more extensive investigation, the same regimen, called the Pritikin Diet, was given to 893 patients (Longevity Research Institute, 1978 b). The patients resided at the study centre for 26 - 30 days and participated in a multimodal program involving diet, exercise, education and medical care. Analysis of the data from the 893 patients indicated significant improvement in risk factors and clinical indices. Mean serum cholesterol and triglyceride levels showed significant reductions,

TABLE 1.9 The effects of dietary fiber[a] upon the plasma lipids

	Plasma cholesterol[b] mg/dl	Plasma triglyceride[b] mg/dl
Study A (1000 mg Cholesterol Diet; 6 subjects[c])		
Fiber-free diet	223 ± 26	102 ± 19
High fiber diet	224 ± 36[d]	83 ± 11[d]
Study B (Cholesterol-Free Diet; 6 subjects[c])		
Fiber-free diet	171 ± 21	103 ± 39
High fiber diet	167 ± 18[d]	93 ± 27[d]

[a] The crude fiber content of the diet was 18 gm/day; the plant cell wall content was 60 gm/day. This comprised a mixture of wheat bran, corn and soybean hulls, cellulose and pectin.

[b] Values are means ± standard error.

[c] Different subjects were enrolled in each of the two separate studies.

[d] Not significant.

SOURCE: Raymond et al., 1977.

as shown in Table 1.10.

Trowell (1977) reported on 20 patients on the Pritikin Diet after 30 days. Results are shown in Table 1.11 and again significant changes in lipid levels were achieved.

A similar high complex carbohydrate low fat diet (HCF) with generous amounts of dietary fiber was given to 13 diabetic men at risk for atherosclerosis (Kiehm et al., 1976). Fasting serum triglyceride and cholesterol levels dropped 15% and 14% respectively after two weeks; a significant reduction over the control American Diabetic Association diet. Further investigations of the HCF diet in 14 diabetic men showed a decrease in serum cholesterol values of 32% over that of controls, and slightly lower values in serum triglycerides (Anderson et al., 1980). Subjects consumed the control diet for 6 - 11 days and the HCF diet for 14 - 35 days. Composition of the diets are shown in Table 1.12. They differed primarily in complex carbohydrate, fiber, total fat and cholesterol.

Lewis (1981) obtained a marked reduction in serum cholesterol levels using diets both modified in fat content and supplemented with fiber (fruit, grains, beans and vegetables). The fat modiffed diet supplemented with fiber contained 27% of energy from fat (P/S ratio = 1.0), 252 mg cholesterol per 2500 kcal and 55 g dietary fiber per 2500 kcal. Twelve Trappist monks were the subjects for this well-controlled study which lasted for five weeks. The results showed a reduction in serum cholesterol and LDL cholesterol of 24 to 29% and 31 to 34% respectively. Serum triglycerides were reduced by 21 to 26%. Lewis suggested that the marked reduction in blood lipids can be attributed

TABLE 1.10 Mean serum lipid changes in 884 patients on the 26 - 30 day longevity centre program[a]

	Range mg/100 ml	Admission mg/100 ml	Discharge mg/100 ml	Percent Change
Cholesterol	≥ 320	380	243	-36%
	300-319	308	211	-36%
	280-299	289	205	-29%
	260-279	269	190	-29%
	240-259	249	186	-25%
	220-239	230	173	-25%
	200-219	211	165	-22%
	180-199	191	152	-20%
	160-179	170	140	-18%
	< 160	145	131	-10%
Average means		235 ± 52	175 ± 37	-26%
Triglycerides	≥ 500	734	223	-70%
	300-499	373	166	-56%
	250-299	274	169	-38%
	200-249	225	155	-31%
	150-199	172	137	-20%
	100-149	124	121	- 2%
	< 100	77	93	+21%
Average means		283	152	-46%

[a] Source: (L.R.I., 1978)

TABLE 1.11 Blood lipid changes in 20 patients after 30 days treatment by diet and exercise (walking)[1]

	Range mg/100 ml	Admission mg/100 ml	Discharge mg/100 ml	Percentage Decrease
Cholesterol	< 200	171	150	12%
	200-224	208	169	19%
	225-249	233	187	20%
	250-274	271	198	27%
	≥ 275	475	208	56%
Triglycerides	110	481	172	64%

[1] Source: Trowell, 1977.

TABLE 1.12 Composition of the high complex carbohydrate, low fat diet (HCF) for the correction[a] of lipoprotein levels in hyperglycemic diabetic men

Component	Control g/day	% of kcal	HCF g/day	% of kcal
Protein	84 ± 3[b]	18%	85 ± 3	18%
Carbohydrate,				
Available (total)	200 ± 8	43%	328 ± 11	70%
Simple	99 ± 4		81 ± 3	
Complex	101 ± 5		247 ± 9	
Fat (Total)	81 ± 3	39%	25 ± 1	12%
Saturated	29 ± 3		6.8 ±.7	
Monounsaturated	35 ± 2		8.5 ±.4	
Polyunsaturated	15 ± 1		7.6 ±.3	
Cholesterol[c]	441 ± 5		46 ± 2	
Plant fiber (total)	20 ±.4		64 ± 2	
Water-soluble	7 ±.5		14 ± 1	
Kcal	1865 ± 72	100%	1876 ± 72	100%

[a] Source: Anderson et al., 1980

[b] ± standard error

[c] mg/day

to the additive effects of two alterations (fat modification and increased fiber intake).

Relatively few patients who develop ischemic heart disease have a single risk factor present in florid form (Anonymous,1975). Far more frequently the increase in risk is due to multiple factors present in moderate degree. In humans, the correction of hyperlipidemia by dietary treatment has shown promise. Many patients have "modestly" elevated cholesterol and triglyceride levels induced by contemporary meal plans (Nash et al., 1977). These levels may not worsen atherosclerosis, but it is possible that lowering them may alleviate it. Nash (1977) reported of 106 patients who showed progression of atherosclerosis on repeat arteriograms. Of these, only one patient had ideal values for both cholesterol and triglyceride. Fifty-one percent had elevated cholesterol levels $\geq$ 250 mg/dl and 55% had elevated triglyceride levels $\geq$ 150 mg/dl. Other risk factors that were also predominant included hypertension (40%), smoking (77%), and a family history of vascular disease (60%).

The following study was designed to provide more information on the effect of dietary treatment on serum lipids and peripheral vascular disease in the long term.

CHAPTER TWO

METHODOLOGY

Methodology

A. Selection of Subjects

Candidates for the study were selected from the medical files of the four major Edmonton hospitals. Each candidate met all of the following criteria:

1. Peripheral vascular disease (PVD) of the aorta or of the iliac, femoral or popliteal arteries, confirmed by arteriography.
2. An independent decision had been made for conservative, as opposed to surgical, treatment.
3. Intermittent claudication of more than one year's duration.
4. Absence or stability of coronary artery disease.
5. No requirement for insulin.
6. Between 45 and 75 years of age.

297 candidates were selected from the 1165 charts reviewed (Table 2.1). Of these candidates, 56 were enrolled in the study. The large number of rejections had several causes: patients were disqualified because of lack of interest, changes in condition, family situation, distance of residence from the project center or medical complications.

The final selection of subjects was based on the following assessments:

1. A physical examination by one of the research project physicians to screen for complicating factors that might have affected the patient's vascular status.

TABLE 2.1 Search for participants

Patient Source	No. of patients reviewed	Candidates selected	No. of subjects entered in study
University of Alberta Hospital (UAH)	500	100	31
Royal Alexandra Hospital	285	58	6
Edmonton General Hospital	180	65	9
Misericordia Hospital	150	49	4
Cardiology Records (UAH)	20	5	-
Health Sciences Records	30	7	-
Referrals (Physician #1)	-	5	2
Referrals (Physician #2)	-	2	2
Referrals (Physician #3)	-	6	2
TOTALS	1,165	297	56

2. Vascular assessment (see Appendix A_1) to determine the severity of the disease.

3. Lifestyle assessment to identify social factors that might affect the patient's ability to follow a diet.

4. Dietary assessment to determine the patient's ability to comply with the diets.

Those agreeing to participation signed an information and consent form (Appendix $B_{1,2,3}$).

Each patient was randomly assigned to one of two groups:

1. American Heart Association (AHA) diet group

2. Pritikin (PRIT) diet group

Each patient was followed for 12 months. Of the 56 subjects who entered the study, 14 did not remain for the entire period for various reasons (Table 2.2). The final group consisted of 42 subjects, with a distribution as shown in Table 2.3.

B. Dietary Groups

The following regimes were used in the study:

1. American Heart Association diet; a diet containing moderate levels of fat of modified composition and moderate levels of fiber. (Subcommittee on Diet and Hyperlipidemia, 1973)

The energy distribution was as follows:

carbohydrate	50-55%
protein	15-20%
fat	25-30%

The P/S ratio was 1.7. The dietary fiber intake was 25-30 grams per 1000 kilocalories (Guidelines appear in Table 2.4).

2. Modified Pritikin diet; a high-fiber, low-fat diet (Longevity Research Institute, 1978b).

TABLE 2.2 Number of withdrawals and reasons for withdrawal

Months Completed	Number of Patients	Reasons for Not Completing Study
12	42	-
6-11	5	Surgery (2)[a] Death (1) Relocation (1) Abandoned diet (1)
2-5	3	Stroke (1) Amputation (1) Insulin required (1)
0-1	6[b]	Death (1) Changed mind (2) Diet too difficult (3)

[a] Number of people withdrawing

[b] Data not used

TABLE 2.3 Number of patients in diet groups

	Number of patients in each group at month		
Group	0	6	12
AHA[a]	23	23	20
M[b]	16	16	15
F[c]	7	7	5
PRIT[d]	27	24	22
M	21	18	18
F	6	6	4

[a] AHA group - moderate fiber, moderate fat diet

[b] M = males

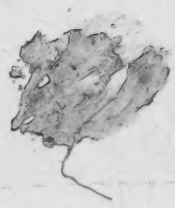
[c] F = females

[d] PRIT group - high fiber, low fat diet

TABLE 2.4 Basic guidelines for diet groups

AHA diet
1. Sugar may only be used in controlled amounts (i.e., diabetic recipes).
2. 100% whole grain breads and cereals must be used.
3. Use lean meats, fish and poultry (maximum 6 oz/day).
4. Use skim milk and skim milk cheese.
5. Use liquid vegetable oils and margarines rich in polyunsaturated fats.
6. Restrict high cholesterol foods; egg yolks must be limited to 3 per week.

PRIT diet
1. No sugars (refined form, i.e., honey, molasses, syrup) may be eaten.
2. 100% whole grain breads and cereals must be used.
3. 1/3 cup raw bran must be taken daily.
4. Use lean meats, fish and poultry (maximum 3 oz/day).
5. Use skim milk and skim milk cheese.
6. No fats or oils may be eaten.
7. No foods high in cholesterol (i.e., eggs, shellfish, may be eaten).



The energy distribution was as follows:

carbohydrate	70-75%
protein	15-20%
fat	5-10%

The dietary fiber intake was 40-45 grams per 1000 kilocalories (Guidelines appear in Table 2.4; P/S ratio was not calculated because the fat intake is extremely low).

Both diets were designed to be nutritionally adequate, meeting the Canadian Dietary Standard (Department of National Health and Welfare, 1976) recommendations for adults 45 to 75 years of age.

The specific composition of the diet was individually tailored for each patient after an interview with the dietitian. A questionnaire was used to collect information about ethnic background, home situation and economic factors which influence food purchases and meal preparation (Appendix C_1). A meal plan was set up for each subject using an exchange system similar to the diabetic food exchange system (Canadian Diabetic Association, 1976). Each subject was assigned a daily quota of exchanges (Table 2.5) for which he could choose foods from various food lists. In this way, the intake of energy, protein, carbohydrate, fat and fiber could be regulated without restricting food choices too much. The subjects were instructed to weigh or measure food portions. They were followed monthly, and calories were adjusted on an individual basis to achieve or maintain ideal weight. The composition of the meal plans and exchange allowances at various energy levels appear in Tables 2.5 to 2.8. Each subject was instructed to have at least three meals per day. Snacks were encouraged, provided that the daily

TABLE 2.5 Daily exchange allowances at different energy levels: AHA diet.

	Calorie Levels					
Exchange group	1000	1200	1500	1800	2000	2500
	(No. of exchanges)					
Meat, Cheese, Eggs	4	5	6	7	8	10
Veg. A	1	2	2	3	3	4
Veg. B	2	2	2	2	2	2
Starches	2	2	3	3	4	4
Breads	3	4	4	5	5	7
Fat	4	5	6	7	8	10
Fruit	3	4	6	8	9	11
Milk	2	2	2	2	2	2

TABLE 2.6 Diet composition at different energy levels: AHA diet

	Calorie Levels					
Diet component	1000	1200	1500	1800	2000	2500
Protein g (% of cals)	50 (20%)	61 (19%)	70 (19%)	81 (18%)	90 (18%)	111 (18%)
Carbohydrate g (%)	131 (52%)	163 (52%)	198 (53%)	240 (54%)	265 (53%)	335 (54%)
Fat g (%)	32 (28%)	40 (29%)	48 (29%)	56 (28%)	64 (29%)	80 (28%)
Fiber g	29	36	44	54	60	76
Sugar/Starch ratio	.8	.8	.9	1.0	1.0	1.0
P/S ratio	1.7	1.7	1.7	1.7	1.7	1.7
Animal/Vegetable protein ratio	2.6	2.4	2.5	2.4	2.3	2.4

TABLE 2.7 Daily exchange allowances at different energy levels: PRIT diet

Exchange group	Calorie Levels (No. of exchanges)					
	1000	1200	1500	1800	2000	2500
Meat, Cheese, Egg Whites	3	3	4	5	6	7
Veg. A	1	2	2	3	3	4
Veg. B	2	2	2	2	2	3
Starches	2	3	4	4	5	6
Legumes (per week)	2	3	3	4	4	5
Breads	6	7	9	11	12	16
Fruit	2	3	4	5	6	6
Bran	1	1	1	1	1	1
Milk	2	2	2	2	2	2

TABLE 2.8 Diet composition at different energy levels: PRIT diet

Diet component	Calorie Levels					
	1000	1200	1500	1800	2000	2500
Protein g (% of cals.)	51 (20%)	57 (18%)	70 (18%)	83 (18%)	94 (18%)	124 (20%)
Carbohydrate g (%)	181 (72%)	228 (75%)	283 (75%)	330 (74%)	370 (74%)	455 (73%)
Fat g (%)	9 (8%)	9 (7%)	12 (7%)	15 (8%)	18 (8%)	21 (7%)
Fiber g	42	54	65	75	83	102
Sugar/Starch ratio	.3	.4	.4	.4	.4	.3
P/S Ratio	(minimal saturated sources)					
Animal/Vegetable protein ratio	1.3	1.0	1.0	1.1	1.1	1.0

quota of exchanges was not exceeded.

All subjects in both groups were given the following guidelines:

1. To exercise 45 minutes a day with a minimum duration of 15 minutes at one time.
2. To restrict alcohol.
3. To restrict cigarettes as much as possible.
4. To restrict caffeine.
5. To avoid adding salt to foods.
6. To use sucaryl (artificial sweetener) in moderation instead of sugar, if necessary.

C. Diet Instruction

Each subject received intensive training on his special diet at the Metabolic Center, University of Alberta Hospital. Seven-hour small group sessions were held daily for four days. The four-day schedule included:

- two diet lectures each day
- one food preparation demonstration
- daily meal planning
- some meal preparation
- two meals and two snacks each day
- blood tests
- two consultations with a physician.

Each subject attended one of these sessions accompanied by his spouse or someone else who prepared meals at his home.

The dietitian followed each patient at monthly intervals. To ensure compliance with the dietary program, each patient completed a

food record for three consecutive days each month and mailed it to the Metabolic Center two weeks before visiting the dietitian. He was asked to record his food intake at home for one weekend day and two week-days. Any problems in following the diet that were revealed by this record were discussed at the next counselling session. The patients were given extensive feedback on changes in weight, blood values and vascular parameters.

D. Assessment

Each patient was assessed on entry into the study and at monthly intervals. Each assessment included one or more of the following (see subsequent sections):

- dietary assessment
- lifestyle assessment
- anthropometric measurements
- biochemical assessment
- clinical evaluation

i. Dietary Assessment

The dietary intake of each patient was assessed at months 0,1, 2,3,4,6,8,10 and 12. Each dietary assessment consisted of two parts:

a) a 3-day food record

b) a 48-hour recall

a) 3-day food record: The patient completed a food record for three consecutive days each month (previously mentioned). These records revealed practical problems in meal planning and were used for counselling, not for collecting data.

b) 48-hour recall: The daily intakes of energy, protein, fat, carbohydrate, dietary fiber and cholesterol were calculated by computer from the results of the 48-hour recall. The dietitian asked the patient to recall all foods and beverages he had consumed in the 48 hours from midnight three days before to midnight the day before the interview. All items were listed in chronological order to minimize the chance of overlooking a food item. The form used for recording appears in Appendix C_2. The technique was based on that used in the Nutrition Canada National Survey (Dept. of National Health and Welfare, 1973a) and was standardized using the guidelines from the Nutrition Canada Survey Resource book (Dept. of National Health and Welfare, 1973b). Guidelines for interviewing and recording data appear in Appendix C_3. One interviewer collected all the data for the study.

The interviewer used food models representing specific volumes to assess the amount of each item consumed. The food model kit, assembled to Nutrition Canada specifications, included blocks of wood, plaster of Paris mounds, glasses and spoons. A description of the food models appears in Appendix C_4.

Each item recorded was later coded using a set of alphanumeric codes with standardized meanings. Coding was done by the dietitian because it requires intimate knowledge of foods and food preparation and of the coding system.

The composition of combination dishes was calculated with information from two publications:

Agriculture Research Service (1966) Procedures for calculating nutritive values of home prepared foods. ARS publ. #62-13.

Adams, C.F. (1975) Nutritive Value of American Foods in Common Units. United States Dept. of Agriculture Handbook No. 456, Govt. Printing Office, Washington, D.C.

The food intake data were keypunched, transferred to computer tape and entered in the computer. Values were calculated for intakes of energy, protein, carbohydrate, fat, dietary fiber, and cholesterol. The food composition data in the computer program were derived from:

1. Watt, B.K. and Merrill, A.L. (1963). Composition of foods - Raw, Processed, Prepared. United States Dept. of Agriculture Handbook No. 8. Govt. Printing Office, Washington, D.C. (values for energy, protein, carbohydrate, fat).

2. Dept. of National Health & Welfare (1973). Nutrition Canada Survey Resource Book. Canadian Food Codes (values for energy, protein, carbohydrate, fat for unique Canadian food items).

3. McCance, R.A. and Widdowson, E.M. (1978). The Composition of Foods, 4th Ed. (Paul, A.A. and Southgate, D.A.T. eds.). Elsevier/North-Holland Biomedical Press (values for dietary fiber).

4. Feeley, R.M., Criner, P.E. and Watt, B.K. (1972). Cholesterol content of foods. J. Am. Diet. Assn. 61, 134 (values for cholesterol).

5. Nutrition Coding Center (N.C.C.), Minneapolis, Minnesota. Cholesterol Values supplied by Lipid Research Clinic, St. Michael's Hospital, Toronto, Canada.

ii. Lifestyle Assessment

The lifestyle questionnaire (Appendix D_1) was used when the dietitian assessed the subject on entry into the study and at each month thereafter. The following items were assessed:

a) exercise - the type of activity
- the duration (minutes)
- frequency (per day or week)

b) tobacco - the average number of cigarettes per day (no patient smoked a cigar or pipe)

c) alcohol consumption - average number of drinks per week
- volume of each drink
- type of alcohol consumed and its percentage of pure alcohol (Watt & Merrill, 1963).

d) caffeine - this was assessed with dietary recalls.

In addition, the motivation and subjective feelings of each subject were discussed.

iii. Anthropometric Measurements

The following anthropometric measurements were collected for each patient:

height (without shoes)
weight (using the same clinical scale each time)
wrist circumference
mid-arm circumference
triceps skinfold thickness
biceps skinfold thickness
intrascapular skinfold thickness
suprailiac skinfold thickness

Lange skinfold calipers were used to measure skinfold thickness. The following calculations were made with the above information.

1. Relative body weight = $\frac{\text{actual weight}}{\text{ideal weight}} \times 100$

Ideal weights were determined from the following information:

a) height
b) frame size (see below)
c) desirable weight tables, prepared by Metropolitan Life Insurance Company (see Appendix D_2)

2. Frame size - based on wrist circumference and height, a frame size of small, medium or large was determined from Body Frame Type tables (Lindner & Lindner, 1973) (see Appendix D_3).

3. Percentage body fat - Lange calipers were used for measurements of skinfold thickness at four sites - biceps, triceps, sub-scapular and suprailiac. Each measurement was done three times and average values were recorded. An average value for all four sites was calculated and compared to percentage body fat tables specific for age and sex (Durnin & Rahaman, 1967) (see Appendix D_4).

This information was recorded on a patient history form (Appendix D_5). Percentage body fat was determined initially and at the end of the study. Relative body weight was determined monthly.

iv. Biochemical Assessment

Biochemical assessment was performed on each subject on entry into the study and at months, 1,2,4,6 and 12. Blood samples were taken by venepuncture at the Metabolic Center, University of Alberta Hospital, after a 12-hour fast. The following blood values were obtained:

- triglyceride
- cholesterol
- high density lipoprotein cholesterol

The biochemical analysis was performed by the Department of Laboratory Medicine, University of Alberta Hospital. Serum cholesterol was determined by the method of Abell and co-workers (1958) using the Liebermann-Burchard reaction. Serum triglyceride was determined on the Abbott bichromatic analyzer (Abbott Diagnostics, California) using the lipase-esterase glycerokinase reaction as described by von Schmidt & von Dahl (1968). HDL was separated from LDL and VLDL by precipitation of the LDL and VLDL with heparin and manganese chloride , according to the method of Burstein et al. (1970).

Cholesterol concentration of the HDL fraction was measured by the cholesterol-esterase/cholesterol oxidase reaction (Klose et al., 1975). Actual analysis of LDL was not done because of the complexity and expense of the procedure. LDL was estimated using the formula: LDL cholesterol = Total cholesterol - HDL cholesterol - 1/5 triglyceride (Friedewald et al., 1972). For careful lipid appraisal, a profile of total cholesterol, HDL-cholesterol, and triglyceride is sufficient in most cases (Kannel et al., 1979; Spritz, 1980).

v. Clinical Assessment

Vascular assessment of each subject was performed initially and at two-month intervals. Measurements included the following:

a) walking distance on a treadmill at a 2% grade and a speed of 1.50 miles per hour (mph) for the first 10 minutes, 2.25 mph for the second 10 minutes, and 3.00 mph for the next 10 minutes. No patient was kept on the treadmill longer than 30 minutes. The treadmill was stopped as soon as claudication occurred.

b) ankle blood pressure before and after reactive hyperemia.

c) analysis of velocity pulse waves from the dorsalis pedis and posterior tibial arteries. These measurements were used to localize any change in arterial disease.

Vascular assessment was performed by the Department of Physiology at the University of Alberta. Extensive detail of this aspect of the study is to be published elsewhere (Oberle, 1982).

E. Statistical Methodology

Various methods were used to analyze the data. Means and standard deviations were determined for the two groups, and for males and females separately within the groups.

The t-test was used to determine if there was a significant difference between groups at month 0. A paired t-test was also used to identify any significant change from month 0 to month 12 for each group.

Pearson correlation coefficients were determined between all variables for each group. Analysis of variance was used to determine whether there was a significant difference between groups in each of the variables at different times during the study. Covariate analysis was used to determine differences in various clinical parameters by group when adjusted for the parameter at month 0.

CHAPTER THREE

RESULTS

A. Description of Groups

Table 3.1 shows the characteristics of the American Heart Association (AHA) group and the Pritikin diet (PRIT) group. Statistical analysis (t-test) revealed that there were no significant differences in the mean ages, weights, heights, relative body weights (RBW) and body fat content of the AHA and PRIT groups for either males or females at entry.

B. Clinical Characteristics

Table 3.2 shows the blood lipid levels and walking times of the AHA and PRIT groups on entry into the study. Statistical analysis revealed that there were no significant differences in the mean values for triglycerides, cholesterol, high density lipoprotein (HDL), low density lipoprotein (LDL), or walking time between AHA and PRIT groups. The mean triglyceride level of the AHA males was influenced by one subject whose value exceeded 10,000 mg/dl.This resulted in a mean value for the AHA group of 607 $\pm$ 2049. In Table 3.2 this subject's values were deleted.

The mean walking times for the males of the AHA and PRIT groups were 9.3 min. and 12.2 min. respectively. The mean walking times for the females of the AHA and PRIT groups were 5.5 min. and 7.7 min. respectively.

C. Lifestyle Habits

Table 3.3 shows the numbers of patients in the various groups who exercised regularly, who smoked cigarettes and who consumed alcohol. There was little difference in lifestyle habits between

TABLE 3.1 Subject characteristics of the AHA and PRIT groups[1]

Group		n	Age	Weight	RBW[2]	Height	Body Fat
			yr	kg	%	cm	%
					means ± SD		
AHA							
	males	16	61±7	80.6±14	111±16	174±5	21±5
	females	7	60±7	67.1±12	117±17	159±5	39±10
PRIT							
	males	21	62±6	76.5±9	108±12	172±4	22±5
	females	6	62±7	62.7±8	111±11	160±3	35±9

[1] No significant difference between groups in all parameters (Student's t-test)

[2] RBW = relative body weight = (actual body weight ÷ ideal body weight) x 100

TABLE 3.2 Blood lipids and walking times at month 0[1]

Parameter	Group					
	AHA			PRIT		
	Total	M	F	Total	M	F
n	23	16	7	27	21	6
	means ± SD					
Serum triglycerides (mg/dl)	180±75[2]	178±77[3]	185±78	236±285	192±53	391±611
Serum cholesterol (mg/dl)	252±71	241±82	275±39	249±46	248±33	253±82
Serum high density lipoproteins (mg/dl)	45±8	43±8	50±5	48±11	46±9	56±13
Serum low density lipoproteins (mg/dl)	153±58	137±61	188±30	159±30	164±30	137±24
Walking times (minutes)	7.9±6.5	9.3±6.9	5.5±5.2	11.3±7.5	12.1±7.5	7.7±7.4

[1] No significant difference between groups in all parameters (Student's t-test).

[2] n = 22 for this value only.

[3] n = 15 for this value only.

TABLE 3.3 Lifestyle habits of the AHA and PRIT groups[1]

Group	Percent of patients who regularly:					
	Exercise[2]		Smoke[3]		Drink[4]	
	n	% of group	n	% of group	n	% of group
AHA	8	35	14	61	9	39
M	5	31	7	44	6	38
F	3	43	7	100	3	43
PRIT	10	37	12	44	8	30
M	7	33	10	48	5	24
F	3	50	2	33	3	50

[1]No significant difference between groups (Student's t-test).

[2]Those who answered "yes" to the question: "Do you exercise regularly?"

[3]Those who smoked $\geq$ 1 cigarette per day.

[4]Those who drank $\geq$ 1 ml absolute alcohol per day.

the groups. For males, the AHA group had a slightly higher percentage of drinkers (38%) than the PRIT group (24%). There were no significant differences in lifestyle habits between groups.

D. Dietary Assessment

Tables 3.4 and 3.5 show the assessment of dietary intakes on entry into the study. Energy intakes (kcal per day) and percentage distribution of energy (percent of total kilocalories from protein, fat and carbohydrate) are given.

Cholesterol intakes for males averaged 330 and 316 mg/day for the AHA and PRIT groups respectively. Cholesterol intakes for females averaged 223 and 166 mg/day for the AHA and PRIT groups respectively.

Dietary fiber intakes for males averaged 16 and 18 gm/day for the AHA and PRIT groups respectively. Dietary fiber intakes for females averaged 21 and 14 gm/day for the AHA and PRIT groups respectively.

Statistical analysis revealed that there was no significant difference in dietary intake between the groups.

E. Effect of Dietary Manipulation

i. Changes in dietary intake

Table 3.6 outlines the dietary plan for the AHA and PRIT groups. Table 3.7 shows the percentage distribution of energy consumed as protein, fat, and carbohydrate at month 0, 6 and 12 of the study. During the study the AHA group essentially met its target of 15-20% of the energy from protein, 25-30% from fat, and 50% from carbohydrate. The PRIT group also came close to its target

TABLE 3.4 Daily intake of dietary components at month 0: males[1]

Dietary Component	Mean Intake: Total Intake		Mean Intake: per kg Body Weight		Total Calories %	
	AHA	PRIT	AHA	PRIT	AHA	PRIT
Kilocalories	1863±669	1992±501	23.4±9.0	27.9±7.5	100	100
Protein(g)	70±23	82±23	.9±.3	1.1±.3	15	17
Fat						
total(g)	87±46	91±33	1.1±.6	1.2±.5	42	41
cholesterol(mg)	330±211	316±166	4.3±3.1	4.2±2.4		
Carbohydrate						
total(g)	200±63	210±50	2.6±1.0	2.8±.7	43	42
dietary fiber(g)	16±8	18±10	.2±.1	.3±.1		

[1]No significant difference between groups (Student's t-test).

TABLE 3.5 Daily intake of dietary components at month 0: females[1]

Dietary Component	Mean Intake: Total Intake		Mean Intake: per kg Body Weight		Total Calories %	
	AHA	PRIT	AHA	PRIT	AHA	PRIT
Kilocalories	1373±389	1099±363	23.4±8.0	27.6±7.6	100	100
Protein(g)	63±10	46±14	1.0±.3	.8±.3	18	17
Fat						
total(g)	51±19	43±17	.8±.4	.7±.4	34	35
cholesterol(mg)	223±78	166±128	3.4±1.3	2.9±2.6		
Carbohydrate						
total(g)	165±58	133±54	1.6±1.3	2.2±1.0	48	48
dietary fiber(g)	21±16	14±7	.3±.1	.3±.1		

[1]No significant difference between groups (Student's t-test).

TABLE 3.6 Dietary plan.

Dietary Component	AHA	PRIT
Kilocalories	100%	100%
Protein (% of kcal)	15-20	15-20
Fat		
% of kcal	25-30	10-15
cholesterol(mg)	< 300	< 100
Carbohydrate		
% of kcal	50-55	70-75
dietary fiber (g/1000 kcal)	25-30	40-45

TABLE 3.7 Energy intake and percentage distribution at months 0, 6 and 12.

Group	n	PRO	FAT	CHO	KCAL
			% of total kcal means		
Month 0					
AHA M	16	15	42	43	1863
F	7	18	34	48	1373
PRIT M	21	17	41	42	1992
F	6	17	35	48	1099
Month 6[1]					
Target values:		(15-20)	(25-30)	(50-55)	
AHA M	16	21	32	47	1487
F	6	21	27	52	1115
Target values:		(15-20)	(10-15)	(70-75)	
PRIT M	21	21	14	65	1605
F	6	22	12	66	1184
Month 12[2]					
Target values:		(15-20)	(25-30)	(50-55)	
AHA M	15	20	29	51	1709
F	5	20	29	51	1341
Target values:		(15-20)	(10-15)	(70-75)	
PRIT M	18	24	14	62	1718
F	6	22	15	63	1386

[1]Mean values of 48 hr. recalls at months 1,2,3,4,6.

[2]Mean values of 48 hr. recalls at months 8,10,12.

distribution, which was very different: 15-20% of the energy from protein, 10-15% from fat and 70-75% from carbohydrate.

Values for month 6 are average intakes assessed by 48-hour recalls at months 1, 2, 3, 4 and 6. Values for month 12 are average intakes of months 8, 10, and 12 (for Tables 3.7 - 3.13).

Tables 3.8 and 3.9 show the average intakes of cholesterol and dietary fiber at months 6 and 12 compared to month 0, and Table 3.10 shows the sources of the dietary fiber.

For the AHA group the average cholesterol intake was 202 mg for males and 149 mg for females at month 6; and 230 mg for males and 173 mg for females at month 12. The target intake for the AHA group was 300 mg or less of dietary cholesterol per day.

For the PRIT group the average cholesterol intake was 105 mg for males and 77 mg for females at month 6; and 127 mg for males and 88 mg for females at month 12. The target for the PRIT group was 100 mg or less cholesterol per day.

For the AHA group the average dietary fiber intake was 15.0 g/1000 kcal for males and 16.4 g/1000 kcal for females at month 6; and 14.6 g/1000 kcal for males and 19.8 g/1000 kcal for females at month 12.

For the PRIT group the average dietary fiber intake was 28.6 g/1000 kcal for males and 30.1 g/1000 kcal for females at month 6; and 27.1 g/1000 kcal for males and 28.6 g/1000 kcal for females at month 12. The PRIT group consumed a higher percentage of fiber from cereals and a lower percentage of fiber from fruits than the

TABLE 3.8 Daily intake of cholesterol at months 0, 6[1] and 12[2].

Group	(n)	Months 0	6[3]	12[4]
			mg (means ± SD)	
AHA			(Target < 300 mg)	
males	16	330±166	202±62	230±123
females	7	223±78	149±37	173±53
PRIT			(Target < 100 mg)	
males	21	316±166	105±39	127±48
females	6	166±128	77±23	88±44

[1]Mean values of 48-hr. recalls at months, 1,2,3,4,6.

[2]Mean values of 48-hrs. recalls at months 8,10,12.

[3,4]Significant difference between groups (analysis of variance $p < 0.05$).

TABLE 3.9 Daily intake of fiber at months 0,6[1] and 12[2].

Group	(n)	Months 0	6[3]	12[4]
			g/1000 kcal (means ±SD)	
AHA			(Target 20-25 g/1000 kcal)	
males	16	8.7±2.6	15.0±3.9	14.6±4.2
females	7	12.2±4.5	16.4±4.3	19.8±2.2
PRIT			(Target 40-45 g/1000 kcal)	
males	21	9.1±4.2	28.6±5.1	27.1±6.0
females	6	13.2±4.7	30.1±8.2	28.6±7.0

[1]Mean values of 48-hr. recalls at months 1,2,3,4,6.

[2]Mean values of 48-hr. recalls at months 8,10,12.

[3,4]Significant difference between groups (analysis of variance $p < 0.001$).

TABLE 3.10 Percent contribution of various sources of fiber at months 0, 6[1] and 12[2].

Group	Fiber source	(n)	Months 0	6	12
				(percentages)	
AHA (total)	cereal	23	54%	44%	50%
	fruit		16%	25%	20%
	veg.		29%	30%	28%
(males)	cereal	16	51%	43%	52%
	fruit		15%	27%	19%
	veg.		31%	29%	27%
(females)	cereal	7	58%	45%	44%
	fruit		16%	20%	24%
	veg.		26%	35%	32%
PRIT (total)	cereal	27	51%	54%	56%
	fruit		12%	16%	19%
	veg.		35%	30%	25%
(males)	cereal	21	54%	55%	57%
	fruit		10%	18%	18%
	veg.		33%	27%	24%
(females)	cereal	6	42%	50%	55%
	fruit		20%	13%	19%
	veg.		37%	37%	26%

[1]Mean values of 48-hr. recalls at months 1,2,3,4,6.

[2]Mean values of 48-hr. recalls at months 8,10,12.

AHA group. However, the PRIT group's total intake of fiber was about twice that of the AHA group.

As shown in the tables, therefore, the food intake of the patients came close to meeting the targets prescribed for the AHA and PRIT groups, and intakes were significantly different between groups for both the 6 and 12 month values.

Tables 3.11 and 3.12 summarize the mean daily intakes of dietary components of the two groups at month 6 and month 12 for the male patients. Tables 3.13 and 3.14 give the same information for the female patients of both groups.

In the males, there were only slight increases in all dietary components between months 6 and 12. Similarly the females of both groups had slight increases in all nutrients between months 6 and 12. For a breakdown of carbohydrate intake as sugars and starches, see Appendix E_1.

ii. Changes in body composition

Table 3.15 shows the body weight, relative body weight and body fat content of patients in the AHA and PRIT groups at 0, 6 and 12 months. The mean weight loss recorded for the male patients was 5.9 kg and 5.1 kg for the AHA and PRIT group, respectively. The weight loss recorded for the female patients was much greater for the PRIT group than the AHA group: 12.8 kg and 1.9 kg respectively. Some of the females could not tolerate the high-fiber Pritikin diet. They experienced flatulence, bloating and abdominal pain and they could not eat enough food to maintain weight.

TABLE 3.11. Daily intake of dietary components at months 6[1] and month 12[2]: males - AliA group.

Dietary component	Intakes					
	totals		per kg wt		% of kilocalories	
months	6	12	6	12	6	12
	means ± SD					
Kilocalories	1487±403	1709±446	20.5±7.5	24.0±8.7	100	100
Protein (g)	75±19	78±22	1.0±0.4	1.1±0.4	21	18
Fat (g)	53±14	65±20	0.8±0.2	0.9±0.4	32	34
Cholesterol (mg)	202±62	230±123	2.8±1.2	3.2±2.0		
Carbohydrate (g)	178±58	204±53	2.5±1.0	2.9±1.1	47	48
Dietary fiber (g)	23±10[3]	24±10[3]	0.3±0.2	0.3±0.1		

[1]Mean values of months 1,2,3,4,6.

[2]Mean values of months 8,10,12.

[3]Significant difference from month 0 (Student's t-test $p < .01$).

TABLE 3.12 Daily intake of dietary components at months 6[1] and month 12[2]: males - PRIT group.

Dietary component	Intakes					
	totals		per kg wt		% of kilocalories	
months	6	12	6	12	6	12
	means ± SD					
Kilocalories	1609±332	1718±339	23.6±5.4	24.2±5.1	100	100
Protein (g)	84±16	98±35	1.2±0.3	1.4±0.5	21	24
Fat (g)	25±10[3a]	28±11[3a]	0.4±0.2	0.4±0.2	14	14
Cholesterol (mg)	105±39[3a]	127±48[3a]	1.5±0.6	1.8±0.7		
Carbohydrate (g)	262±61[3b]	269±59[3b]	3.8±0.9	3.8±1.0	65	62
Dietary fiber (g)	44±13[3a]	44±12[3a]	0.6±0.2	0.6±0.2		

[1]Mean values of months 1,2,3,4,6.

[2]Mean values of months 8,10,12.

[3]Significant difference from month 0 (Student's t-test) a($p < 0.001$) b($p < 0.05$).

NOTE: Values for PRIT males are significantly different than for AHA males in carbohydrate ($p < 0.01$), fat ($p < 0.01$), cholesterol ($p < 0.05$) and fiber ($p < 0.001$) - analysis of variance.

TABLE 3.13 Daily intake of dietary components at month 6[1] and month 12[2]: females - AHA group.

Dietary component[3]	Intakes					
	totals		per kg wt		% of kilocalories	
months	6	12	6	12	6	12
	means ± SD					
Kilocalories	1115±144	1341±256	18.2±4.0	21.3±7.0	100	100
Protein (g)	59±9	65±10	1.0±0.2	1.0±0.3	21	20
Fat (g)	33±6	45±16	0.6±0.2	0.7±0.3	27	29
Cholesterol (mg)	149±37	173±53	2.4±0.6	2.7±1.0		
Carbohydrate (g)	145±28	170±34	2.4±0.6	2.7±0.9	52	51
Dietary fiber (g)	18±6	23±3	0.3±0.1	0.4±0.1		

[1]Mean values of months 1,2,3,4,6.

[2]Mean values of months 8,10,12.

[3]No significant change in any dietary component from month 0 (Student's t-test).

TABLE 3.14 Daily intake of dietary components at month 6[1] and month 12[2]: females - PRIT group.

Dietary component[3]	Intakes					
	totals		per kg body wt.		% of kilocalories	
months	6	12	6	12	6	12
	means ± SD					
Kilocalories	1184±269	1346±439	22.4±1.0	32.5±8.9	100	100
Protein (g)	64±14	76±21	1.3±0.3	1.7±0.5	22	22
Fat (g)	17±7[3a]	23±11[3a]	0.7±0.9	0.5±0.2	12	15
Cholesterol (mg)	77±23[3a]	88±44[3a]	1.8±1.0	1.0±1.1		
Carbohydrate (g)	195±44[3b]	219±77[3b]	3.9±1.2	5.3±1.2	66	63
Dietary fiber (g)	35±12[3a]	40±12[3a]	0.7±17	0.9±0.3		

[1] Mean values of months 1,2,3,4,6.

[2] Mean values of months 8,10,12.

[3] Significant difference from month 0 (Student's t-test) a($p < 0.001$) b($p < 0.05$).

TABLE 3.15 Changes in body weight (kg), relative body weight (RBW) and body fat (%) at 6 and 12 months.

Weight parameter		AHA		PRIT	
		M	F	M	F
		means ± SD			
Body weight	mo. 0	80.6±14	67.1±12	76.5±9	62.7±8
	mo. 6[1]	75.3±13	62.7±11	70.1±8	55.2±8
	mo. 12	74.7±13	65.2±10	71.4±6	49.9±5
	% change[2]	-7%	-4%	-7%	-19%
Relative body weight	mo. 0	111±15	117±17	108±12	111±11
	mo. 6	103±14	110±16	99±8	97±12
	mo. 12	103±15	112±17	101±8	90±8
	% change	-7%	-4%	-7%	-19%
Body fat	mo. 0	21±5	39±10	22±5	35±9
	mo. 12[3]	20±5	35±10	19±3	30±10
	% change	-5%	-10%	-14%	-11%

[1] Significant difference between groups at month 6 only. ($p < 0.01$)- analysis of variance.

[2] Change from month 0.

[3] Values not determined at month 6.

iii. Changes in blood glucose

Although glucose values were not the primary concern of this investigation, Table 3.16 has been included as additional information. PRIT males showed the best response with a 17% decrease in fasting glucose values at month 6, which was maintained until month 12. AHA males had a 9% decrease at month 6, but then a 4% increase at month 12. PRIT females showed decreases of 8% at month 6, and a further decrease of 9% at month 12. AHA females had a 5% decrease at month 6, but then a 4% increase at month 12.

iv. Changes in blood lipids

Blood levels at month 0, 6 and 12 are shown for triglycerides (Table 3.17); for cholesterol (Table 3.18); for high density lipoprotein (HDL) (Table 3.19); and for low density lipoprotein (LDL) (calculated values) (Table 3.20). There was no significant difference between the AHA and PRIT groups for any of these variables at any point in the study, with the exception of cholesterol at month 4 ($p < 0.01$ analysis of co-variance). Levels of triglycerides, cholesterol, and LDL for males and females in both groups decreased throughout the study. Levels of HDL increased throughout the study except for PRIT females in the second six months. See Table 3.38 for significant changes within groups.

The patients in the AHA and PRIT groups were subdivided according to whether they lost more or less than 5 kg body weight in 12 months.

Table 3.21 shows percentage change in serum triglyceride levels and serum cholesterol levels of AHA and PRIT patient

TABLE 3.16 Serum fasting[1] glucose values at months 0, 6 and 12[2].

Group	(n)	Month 0	Month 6		Month 12	
			concentration	change[3]	concentration	change[4]
		mg/dl	mg/dl	%	mg/dl	%
				means ± SD		
AHA						
males	(16)	108±26	98±23	-9	102±29	+4
females	(7)	105±18	100±19	-5	104±21	+4
PRIT						
males	(21)	105±55	87±9	-17*	88±9	+1
females	(6)	97±17	90±17	-8	82±27	-9

[1] 12 hour fast (overnight).

[2] Average values of months 2, 4 and 6.

[3] % change from month 0.

[4] % change from month 6.

* Significant difference between group at Month 6 only (analysis of co-variance).

TABLE 3.17 Serum triglyceride values at months 0, 6 and 12.

Group	(n)	Month 0	Month 6[1]		Month 12	
			concentration	change[2]	concentration	change[3]
		mg/dl	mg/dl	%	mg/dl	%
			means ± SD			
AHA						
males	(15)	178±77	157±81	-12%	209±206	+33%
females	(7)	185±78	170±76	-8%	193±113	+14%
PRIT						
males	(21)	192±53	164±47	-15%	167±65	+2%
females	(6)	391±611	270±355	-31%	112±47	-59%

[1] Mean values of months 2, 4 and 6.

[2] % change from month 0.

[3] % change from month 6.

NOTE: No significant difference between groups (analysis of co-variance).

NOTE: The data of all lipid values for each month is shown in Appendix E_4.

TABLE 3.18 Serum cholesterol values at months 0, 6 and 12.

Group	(n)	Month 0	Month 6[1]		Month 12	
			concentration	change[2]	concentration	change[3]
		mg/dl	mg/dl	%	mg/dl	%
			means ± SD			
AHA						
males	(16)	241±82	211±61	-13%	222±61	+5%
females	(7)	275±39	267±58	-3%	268±52	-
PRIT						
males	(21)	248±33	212±32	-15%	229±40	+8%
females	(6)	253±82	230±57	-9%	202±25	-12%

[1] Mean values of month 2, 4 and 6.

[2] % change from month 0.

[3] % change from month 6.

NOTE: No significant difference between groups (analysis of co-variance).

TABLE 3.19 Serum HDL values at months 0, 6 and 12.

Group	(n)	Month 0	Month 6[1]		Month 12	
			concentration	change[2]	concentration	change[3]
		mg/dl	mg/dl	%	mg/dl	%
			means ± SD			
AHA						
males	(16)	43±8	45±11	+5%	47±9	+4%
females	(7)	50±5	52±4	+4%	58±10	+12%
PRIT						
males	(21)	46±9	45±9	-2%	49±11	+9%
females	(6)	56±13	55±14	-2%	54±9	-2%

[1] Mean values of months 2, 4 and 6.

[2] % change from month 0.

[3] % change from month 6.

NOTE: No significant difference between groups (analysis of co-variance).

TABLE 3.20 Serum LDL[1] at months 0, 6 and 12.

Group	(n)	Month 0	Month 6[2]		Month 12	
			concentration	change[3]	concentration	change[4]
		mg/dl	mg/dl	%	mg/dl	%
			means ± SD			
AHA						
males	(16)	137±61	113±26	-18%	134±37	+19%
females	(7)	188±30	191±42	+2%	171±39	-11%
PRIT						
males	(21)	164±30	134±27	-18%	149±34	+11%
females	(6)	137±24	122±15	-11%	125±22	+3%

[1] LDL values determined by formula: CHOL-HDL-1/5 TG.

[2] Mean values of months 2, 4 and 6.

[3] % change from month 0.

[4] % change from month 6.

NOTE: No significant difference between groups (analysis of co-variance).

TABLE 3.21 Percentage change in serum triglyceride and serum cholesterol levels after one year with weight loss[1].

	Weight loss < 5.0 kg		Weight loss ≥ 5.0 kg	
	n	Δ	n	Δ
% Change in TG				
AHA				
males	8	+6%	7	-21%
females	5	-4%	2	+33%
PRIT				
males	11	+3%	10	-29%
females	1	+1%	5	-21%
% Change in CHOL.				
AHA				
males	8	-2%	7	-4%
females	5	-4%	2	-5%
PRIT				
males	11	-2%	10	-16%
females	1	-2%	5	-6%

[1] Consistently lower lipid values with weight loss (see correlations Table 3.35).

groups by weight loss of greater or less than 5 kg. The patients in both study groups who lost more than 5 kg of body weight in 12 months had a greater decrease in serum triglyceride and cholesterol levels than those who lost less than 5 kg. The male Pritikin patients with weight loss over 5 kg had the greatest decrease in both triglycerides and cholesterol.

Table 3.22 shows percentage change in the HDL and LDL lipoproteins of AHA and PRIT patients grouped by weight loss of greater or less than 5 kg. The patients who lost more than 5 kg of body weight in 12 months had a greater increase in serum HDL and decrease in serum LDL levels than those who lost less than 5 kg.

The HDL levels of male non-drinkers were compared with those of moderate drinkers (7-15 ml absolute alcohol per day or 5-6 oz. of alcohol per week) to test Castelli's hypothesis (1977) that moderate drinkers have 10% higher HDL levels than non-drinkers. Table 3.23 compares the HDL levels of the males classified as moderate drinkers and non-drinkers.

The moderate drinkers had higher HDL levels than non-drinkers in both the male AHA group and PRIT group. For the total male group (both AHA and PRIT subjects) the HDL levels of the moderate drinkers were 10% higher than those of the non-drinkers at month 0 and 17% higher at month 12. This is in agreement with Castelli's findings, but the differences between non-drinkers and drinkers were not significant, perhaps because of the small numbers investigated.

The effect of smoking on serum cholesterol levels was examined. Table 3.24 compares the serum cholesterol levels of patients

TABLE 3.22 Percentage change in lipoprotein levels after one year with weight loss[1].

	Weight loss < 5.0 kg		Weight loss ≥ 5.0 kg	
	n	Δ	n	Δ
% Change in HDL				
AHA				
males	8	+5%	7	+11%
females	5	-5%	2	+43%
PRIT				
males	11	+8%	10	+6%
females	0	-	5	+9%
% Change IN LDL				
AHA				
males	8	-4%	7	-3%
females	5	-2%	2	-24%
PRIT				
males	11	-6%	10	-18%
females	0	-	5	-12%

[1] A trend toward higher HDL and lower LDL values with weight loss.

TABLE 3.23 HDL values of males: non-drinkers[1] compared to moderate drinkers[2].

Group	HDL values (mg/dl) Non-drinkers		Drinkers		% difference
		mean values			
Month 0	(n)		(n)		
Total	(11)	41 [3]	(10)	45 [3]	+ 10%
AHA	(6)	43	(3)	45	+ 5%
PRIT	(5)	38	(7)	45	+ 18%
Month 12					
Total	(12)	46 [3]	(9)	54 [3]	+ 17%
AHA	(6)	48	(2)	53	+ 10%
PRIT	(6)	44	(7)	54	+ 23%

[1] ≤ 1 ml absolute alcohol/day.

[2] 7-15 ml absolute alcohol/day.

[3] No significant difference.

TABLE 3.24 Serum cholesterol values: smokers[1] compared with non-smokers[2].

Group	Cholesterol values (mg/dl) Non-smokers		Smokers		% Difference
		mean values			
Month 0	(n)		(n)		
Total	(26)	253	(21)	251	-1%[3]
Males	(17)	246	(18)	244	-1%
Females	(9)	266	(3)	293	+10%
Month 12					
Total	(21)	225	(7)	241	+6%[3]
Males	(15)	218	(6)	233	+6%
Females	(6)	244	(1)	292	+16%

[1] 0 cigarettes/day.

[2] ≥ 15 cigarettes/day.

[3] No significant difference.

classified as non-smokers and smokers (15 or more cigarettes per day) at entry and completion of the study. There was no difference in serum cholesterol levels between the two groups.

Table 3.25 compares the HDL levels of patients classified as non-smokers and smokers on entry into the study. There was no difference in serum HDL levels between the two groups. For the females, HDL appeared to be much different in smokers (17% lower) but this value only represented one subject and is of little validity.

In both tables (3.24, 3.25) there was a trend which showed the females having consistently higher cholesterol and HDL values than the males regardless of whether they did or did not smoke.

v) Changes in walking ability

Walking time on a treadmill was used as a measure of functional improvement. Table 3.26 presents the walking times of AHA and PRIT groups at months 0, 6 and 12 months. There was a marked improvement in walking times for all groups.

The mean walking times at month 12 ranged from 18.7 min. to 24.3 minutes. The mean walking times for the PRIT group were over 20 minutes; those for the AHA group were less than 20 minutes. At 10 and 20 minutes the speed of the treadmill was increased, so the patients were working harder per minute when they exceeded these levels.

It was found through analysis of variance that there was a significant difference between the two male groups at month 6 ($p < 0.05$) and a trend for the PRIT group males to have better walking times at month 12. The latter was not a significant

TABLE 3.25 Serum HDL values: smokers[1] compared with non-smokers[2].

Group	HDL values (mg/dl)[3] Non-smokers		Smokers		% difference
		mean values			
Month 0	(n)		(n)		
Total	(26)	48	(16)	44	-8%[3]
Males	(17)	45	(15)	44	-2%
Females	(9)	54	(1)	45	-17%
Month 12					
Total	(21)	50	(7)	50	-[3]
Males	(15)	47	(6)	47	-
Females	(6)	57	(1)	69	+17%

[1] 0 cigarettes/day.

[2] ≥ 15 cigarettes/day.

[3] No significant difference.

TABLE 3.26 Walking times at months 0, 6 and 12.

Group	(n)	Month 0	Month 6		Month 12	
			time	change[1]	time	change[2]
		min	min	%	min	%
			means ± SD			
AHA						
males	(13)	9.3±6.9	15.3±7.4	+65%	19.0±6.3	+24%
females	(7)	5.5±5.2	19.1±3.6	+247%	18.7±6.8	~ -2%
PRIT						
males	(20)	12.2±7.5	23.0±7.5[3]	+89%	23.7±7.7	+3%
females	(5)	7.7±7.4	17.7±9.6	+130%	24.3±7.6	+37%

[1] % change from month 0.

[2] % change from month 6.

[3] Significant difference in males only at month 6 ($p < 0.05$ - analysis of variance).

NOTE: Significant difference when males and females combined (analysis of co-variance), at month 4 only.

difference, however, likely because of the large improvement in the AHA males during the second half of the study (24% versus only 3% for the PRIT males).

Table 3.27 and 3.28 show the individual values for walking distance of the subjects at each assessment. There was a significant difference between groups only at month 6 (analysis of covariance $p < 0.05$). It should be noted, however, that 11 patients from the PRIT group walked the maximum distance tested, 1811 meters, while only one AHA subject achieved this distance.

Table 3.29 shows the individual results (dietary and clinical parameters) of 7 male subjects who showed the greatest improvement in walking time (225% or more of an increase in walking time).

Table 3.30 shows the individual results of seven male subjects who showed the least improvement in walking time (less than 30% of an increase in walking time). Again, only males were considered because in many of the other variables, males of both groups showed a similar response.

Table 3.31 compares the mean values of various parameters of the male subjects who showed the greatest and least improvement in walking times. The values showing a difference were:

triglyceride level - (176 mg/dl and 147 mg/dl for the most and least improved)

HDL level - (51 mg/dl and 46 mg/dl for the most and least improved)

LDL level -(123 mg/dl and 136 mg/dl for the most and least improved)

TABLE 3.27 Walking distances (meters)[a] - AHA Group.

Subject (sex)	Subject (no)	Months 0	2	4	6	12
M	04	270	629	437	523	523
M	05	166	433	825	1308	1288
F	08	94	335	1006	1053	1529
M	09	138	211	221	443	1006
F	10	55	70	64	885	---
M	11	845	1093	1247	1006	1167
M	13	--	272	463	623	1006
M	14	144	463	428	448	704
M	23	221	258	1006	558	--
F	29	262	583	674	1006	1207
M	32	1233	1408	1006	1811*	1811*
M	35	84	128	188	231	493
F	37	538	523	583	764	614
M	38	579	402	533	553	523
M	42	994	1140	1127	1167	1408
F	47	30	60	57	--	--
M	49	463	860	--	--	1006
F	50	618	774	885	704	433
F	53	168	201	724	1469	1167
M	55	282	315	--	--	--

*30 min (1811 meters) - maximum time allotted.

[a]Walking distance (WD) = (for first 10 min) = 40.23 m/min x time (min)
(for second 10 min) = 402.3 m/min +
((time(min)-10)x 60.35)
(for third 10 min) = 1005.8 m/min +((time
(min)-20) 80.47)

TABLE 3.28 Walking distances (meters) - PRIT Group

Subject (sex)	(no)	Months 0	2	4	6	12
F	01	39	49	101	180	--
M	02	111	200	1489	1093	1408
F	06	81	404	1167	1569	1811*
M	07	655	1175	1247	1167	1811*
M	15	181	272	389	402	362
M	16	672	885	845	1167	1811*
M	17	30	101	126	191	523
M	18	855	1167	1093	1811*	1811*
F	20	855	1086	1086	907	644
M	21	77	87	81	--	--
M	22	1616	1382	1167	1576	1811*
F	24	278	483	583	598	1247
F	25	548	1362	1422	1489	1811*
M	26	855	946	855	825	523
M	28	1026	1006	1046	1247	1207
M	33	1086	1368	1535	1811*	1811*
M	36	284	275	--	--	523
M	39	329	523	852	1046	1086
M	40	352	201	265	--	--
M	43	1093	1247	1811*	1811*	1811*
M	46	371	1442	1811*	1811*	1811*
M	48	540	614	1086	1167	1247
M	51	848	1362	1811*	1811*	1811*
M	52	324	855	1569	1247	1060
M	54	751	1086	1811*	1811*	1811*

*30 min (1811 meters) - maximum time allotted.

TABLE 3.29 Individual clinical and dietary data on 7 male subjects who showed the greatest improvement in walking time (≥ + 225%).

Subject No	Group	WT[1] response	Body wt change	RBW (Mo 12)	Serum triglyceride conc[2]	Serum triglyceride Δ[3]	Serum cholesterol conc	Serum cholesterol Δ	Serum HDL conc	Serum HDL Δ	Serum LDL conc	Serum LDL Δ
		%	kg	%	mg/dl	%	mg/dl	%	mg/dl	%	mg/dl	%
46	PRIT	+225	-.4	111	321	+69	300	+10	55	+19	179	-4
14	AHA	+319	+1.1	82	162	+30	190	+44	62	+24	96	+68
35	AHA	+452	-7.1	94	108	-37	185	+9	40	+18	123	+21
5	AHA	+469	-10.1	108	190	-39	207	-17	56	-5	113	-11
9	AHA	+485	-11.1	119	111	-20	188	-7	47	+2	119	-8
2	PRIT	+809	-2.9	96	169	-1	142	-5	30	-	84	-2
17	PRIT	+1500	-4.3	93	169	+8	247	-9	66	-2	147	-16

[1] Walking time

[2] Concentration

[3] Δ = change (% difference after 12 months)

TABLE 3.29 (Continued)

Subject No	Dietary Intakes[1] cholesterol	fiber	pro	fat	cho	Lifestyle Habits[2] Smoking	Alcohol			Exercise
	mg	g	%	%	%	pack yrs	cigs/day[3]	[4]	time[5]	frequency[6]
46	204	42	19	21	60	40	2	3	1	2
14	123	18	17	28	55	20	1	1	1	3
35	397	35	19	38	43	0	1	1	2	3
5	164	22	18	34	48	85	1	3	1	1
9	192	26	25	24	51	32	1	1	3	3
2	142	67	22	12	66	13	1	1	4	3
17	97	53	21	13	66	42	1	1	1	3

[1]Averages of months 7-12.

[2]At month 12.

[3]1 0; 2 < 20; 3 ≥ 20 cigarettes per day.

[4]1 ≤ 1; 2 2-6; 3 7-15; 4 16-39; 5 ≥ 40 ml absolute alcohol/week.

[5]1 0-15; 2 16-30; 3 31-45; 4 ≥ 45 minutes.

[6]1 0-2; 2 3-4; 3 5-7; 4 > 7 times per week.

TABLE 3.30 Individual clinical and dietary data on 7 male subjects who showed the least improvement in walking times (< +30%).

Subject No	Group	WT[1] response	Body wt Change	RBW (Mo 12)	Serum triglyceride		Serum cholesterol		Serum HDL		Serum LDL	
					conc[2]	Δ[3]	conc	Δ	conc	Δ	conc	Δ
		%	kg	%	mg/dl	%	mg/dl	%	mg/dl	%	mg/dl	%
26	PRIT	-31	-11.0	114	186	-28	237	-3	41	+11	159	+3
38	AHA	-7	-8.2	89	96	-4	181	-8	58	+32	104	-22
23	AHA	+6	+0.2	84	78	-24	225	+15	42	-9	42	-9
22	PRIT	+9	-0.7	95	126	-34	184	-16	47	+24	112	-22
28	PRIT	+11	-3.1	94	112	-22	236	-2	51	-4	163	+3
42	AHA	+26	-2.3	107	139	-14	190	-23	43	--	119	-30
11	AHA	+27	-2.6	114	293	+74	225	-16	37	-20	129	-32

[1] Walking time

[2] Concentration

[3] Δ = change (% difference after 12 months).

TABLE 3.30 (continued)

Subject No	Dietary intakes[1] cholesterol	fiber	pro	fat	cho	Lifestyle habits[2] smoking	alcohol		exercise	
	mg	g	%	%	%	pack yrs	cigs/day[3]		[4] time[5]	fre[illegible]ncy[6]
26	93	36	20	16	64	20	1	1	2	3
38	575	19	19	38	43	75	2	1	1	3
23	188	31	16	35	49	99	2	1	2	3
22	107	56	20	14	66	60	3	3	2	2
28	101	44	22	9	69	54	3	3	1	3
42	216	39	18	28	54	37	1	4	2	3
11	177	13	17	35	48	40	1	3	1	2

[1] Average of months 7-12.

[2] At month 12.

[3] 1 0; 2 < 20; 3 ≥ 20 cigarettes/day.

[4] 1 ≤ 1; 2 2-6; 3 7-15; 4 16-39; 5 ≥ 40ml absolute alcohol/week.

[5] 1 0-15; 2 16-30; 3 31-45; 4 ≥ 45 minutes.

[6] 1 0-2; 2 3-4; 3 5-7; 4 > 7 times per week.

TABLE 3.31 Comparison of data on subjects who showed the greatest and least improvement in walking times.

Parameter[1]	Groups: Most Improved (n=7) 3 PRIT; 4 AHA	Least Improved (n=7) 3 PRIT; 4 AHA
WT response (%)	+608	+6
Body wt. change (kg)	-5.1	-4.0
RBW (%)	100	100
TG (mg/dl)	176 (+1)[2]	147 (-7)[2]
CHOL (mg/dl)	208 (+4)	211 (-8)
HDL (mg/dl)	51 (+8)	46 (+5)
LDL (mg/dl)	123 (+7)	136 (-10)
D CHOL (mg)	188	208
D F (g)	38	34
PRO (%)	20	19
FAT (%)	24	25
CHO (%)	56	56
Pack Years (yr)	33	55
Cigarettes/day[3]	1.1	1.9
Alcohol[3]	1.6	2.3
Exercise: time[3]	1.9	1.6
frequency	2.6	2.7

[1]Mean values.

[2]% change after one year.

[3]Similar coding as in Tables 3.29 and 3.30.

dietary cholesterol - (188 mg and 208 mg for the most and least improved)

smoking (pack years) - (33 and 55 for the most and least improved

alcohol value - (1.6 and 2.3 for the most and least improved).

It should be noted that the two males that showed the most dramatic improvements (increases of 809% and 1500%) were on the PRIT diet. Subjects number 2 and number 17, as shown in Table 3.29 had very dissimilar biochemical profiles; however, factors that were similar between these two men were that they were slightly underweight at 96% RBW (#2) and 93% RBW (#17); they had high fiber (67 g and 53 g/day) and low fat intakes (12% and 13%/day); and they had quit smoking, quit drinking and were exercising regularly.

One biochemical parameter which may have some association with functional walking improvement is the LDL/HDL ratios. Table 3.32 shows mean LDL/HDL ratios at month 0 and 12. Decreases in the ratios of 10-12% were seen in both groups. Table 3.33 compares mean ratios at various levels of improvement in walking distance. Where there was an improvement in walking ability, as the degree of improvement increased, the mean LDL/HDL ratios decreased in both groups. Four subjects whose walking distances did not improve were an exception to this trend in that they did not have particularly high ratios. Individual values are listed in Appendix E_2.

Table 3.34 shows subjects whose Type II or IV HLP was normalized during the study and the concurrent change in walking distance and LDL/HDL ratios. In these subjects, WD changes were above average and ratios were below average.

TABLE 3.32 Mean LDL/HDL ratios at months 0 and 12.

Group	(n)	Mo. 0	Mo. 12	
AHA all	(16)	3.1	2.8	(-10%)[1]
M	(11)	2.9	2.6	(-10%)
F	(5)	3.7	3.1	(-16%)
PRIT all	(22)	3.4	3.0	(-12%)
M	(18)	3.6	3.1	(-14%)
F	(4)	2.6	2.5	(4%)

[1] % change from month 0.

TABLE 3.33 Mean changes in walking distance after one year and mean LDL/HDL ratios at month 12.

Range (WD) (m)	AHA (n)	AHA WD change (m)	AHA LDL/HDL	PRIT (n)	PRIT WD change (m)	PRIT LDL/HDL
> 750	(6)	+1031	2.5	(11)	+1157	2.9
251-750	(7)	+486	2.8	(5)	+676	3.0
0-250	(1)	+76	4.2	(4)	+199	3.4
< 0	(2)	-121	2.7	(2)	-287	3.0
mean total		+532	3.1		+656	3.0

TABLE 3.34 Changes in walking distance and LDL/HDL ratios at month 12 in subjects whose HLP was normalized after one year.

Subject no	sex	HLP	WD change(m)	LDL/HDL (mo 12)	Subject no	sex	HLP	WD change(m)	LDL/HDL (mo 12)
08	F	IV	+1435	1.9	07	M	IV	+1156	3.8
13	M	IV	+734	1.8	16	M	IV	+1139	1.2
32	M	IV	+578	2.3	22	M	IV	+195	2.4
35	M	IV	+409	3.1	39	M	IV	+757	2.9
42	M	IV	+817	2.8	51	M	II	+963	3.9
mean total			+795	2.4				+842	2.8

Table 3.35 shows significant correlations between major dietary, biochemical and vascular parameters. Pearson correlation co-efficients were used; only those correlations with a significance level of $p < 0.01$ appear in the table. Correlations appearing more than once are not likely to occur by chance. Only parameters measured at the same month were compared.

Serum cholesterol and dietary fiber were negatively correlated at months 2, 4 and 12. Walking time and dietary fiber were correlated at month 4 only. Serum cholesterol was negatively correlated with carbohydrate intake at month 2,4,6 and 12, and with energy intake at months 4 and 6. Relative body weight was correlated with serum cholesterol at month 2 and 6 and with serum triglyceride at month 2,4 and 6. Relative body weight was negatively correlated with HDL at months 2 and 4.

vi. Results compared with Pritikin Research Center results

Table 3.36 compares the effect of the AHA diet and the Pritikin diet on serum triglyceride levels at month 1 with reported results from Pritikin's Longevity Research Institute (L.R.I.). The distribution of serum triglyceride levels is shown. The mean percentage reduction in serum triglyceride levels was greater in the PRIT group of the present study (-35%) than at the L.R.I. (-25%) or with the AHA diet (-23%). There was a large difference in the number of subjects being compared, however.

Table 3.37 compares the effect of the AHA diet and the Pritikin diet on serum cholesterol levels at month 1 with reported results from the L.R.I. The distribution of serum cholesterol

TABLE 3.35 Relationships between clinical and dietary indices.

Dietary Variable	Clinical Variable	Correlation value	Level of Significance
Fat-2[1]	WT-2	-.3546[2] (-.5284 +.0019)[3]	.008
% Fat intake-12	Glucose-12 (2 hr)	+.4059 (+.1238 +.3924	.004
Carbohydrate-2 (CHO)	CHOL-2	-.3914 (-.2927 -.5854)	.002
CHO-4	CHOL-4	-.4049 (-.5579 -.2640)	.002
CHO-4	WT-4	+.4457 (+.0644 +.4237)	.002
CHO-6	CHOL-6	-.3884 (-.4297 -.5055)	.004
CHO-12	CHOL-12	-.4642 (.6033 -.2182)	.001
CHO-12	TG-12	-.3907 (-.5697 +.2638)	.005
Kcal/kg-4	CHOL-4	-.4054 (-.5292 -.1945)	.002
Kcal/kg-6	CHOL-6	-.3702 (.4574 -.3031)	.006
RBW-2	TG-2	+.3617 (+.3903 +.2337)	.005
RBW-2	CHOL-2	+3405 (+.3830 +.2395)	.008
RBW-2	Glucose-2 (2 hrs)	+.3354 (+.4190 +.0715)	.009
RBW-2	HDL-2	-.3608 (-.4583 -.3408)	.005

cont...

TABLE 3.35 Continued

RBW-4	TG-4	+.3753 (+.3961 +.4165)	.004
RBW-4	HDL-4	-.4081 (-.5206 -.3064)	.003
RBW-6	TG-6	+.3452 (+.5085 +.4202)	.009
RBW-6	CHOL-6	+.4421 (+.5627 +.4256)	.001
RBW-6	Glucose-6 (fasting)	+.4456 (+.3376 +.5016)	.001
RBW-6	Glucose-6 (2 hrs)	+.4450 (+.4127 +.3979)	.001
RBW-12	Glucose-12 (fast)	+.3867 (+.3456 +.2989)	.006
Dietary fiber-2 (D F)	CHOL-2	-.3387 (-.2568 -.5435)	.008
DF-4	CHOL-4	-.4194 (-.3130 -.1782)	.001
DF-4	WT-4	+.4766 (+.1422 +.4430)	.001
DF-12	CHOL-12	-.3557 (-.4164 -.3757)	.010
Exercise-12	TG-12	-.4443 (-.5273 -.4605)	.002
Alcohol-12	Smoking-12	+.3617 (+.2287 +.4603)	.005

[1]Month of study.

[2]Pearson correlation co-efficient.

[3]Correlation co-efficient for AHA and PRIT groups respectively.

TABLE 3.36 Comparison of serum triglyceride values after one month with Longevity Research Institute (LRI)[1].

Range mg/dl	Mean Triglyceride Values							
	LRI PRIT[2]		U of A PRIT			U of A AHA		
months	0	1	0	1	(n)	0	1	(n)
< 100	77 -	93	86 -	106	(2)	-		
100 - 150	194 -	121	135 -	131	(7)	117 -	108	(6)
151 - 200	172 -	137	173 -	140	(8)	171 -	159	(10)
201 - 250	225 -	155	235 -	154	(4)	218 -	171	(2)
251 - 300	274 -	169	269 -	180	(3)	-		
301 - 500	373 -	166	302 -	202	(1)	342 -	236	(3)
> 500	734 -	223	1630 -	447	(1)	-		
Mean	174	130	238	157		185	143	
% change		-25%		-35%			-23%	

[1]Longevity Research Institute (LRI, 1978).

[2]n = 881 (distribution was unavailable).

TABLE 3.37 Comparison of serum cholesterol values after one month with Longevity Research Institute (LRI)[1].

Range mg/dl	LRI PRIT[2]	Mean Cholesterol Values U of A PRIT			U of A AHA		
months	0 1	0	1	(n)	0	1	(n)
< 160	145 - 131	150 -	137	(1)	132 -	215	(1)
160 - 180	170 - 140	175 -	181	(1)	170 -	176	(1)
181 - 200	191 - 152	-			197 -	175	(3)
201 - 220	211 - 165	216 -	199	(6)	208 -	176	(4)
221 - 240	230 - 173	229 -	191	(1)	228 -	166	(1)
241 - 260	249 - 186	249 -	198	(9)	249 -	213	(3)
261 - 180	169 - 190	274 -	221	(4)	267 -	233	(4)
281 - 300	289 - 205	291 -	207	(3)			
301 - 320	308 - 211	-			301 -	280	(1)
> 320	380 - 243	409 -	311	(1)	374 -	320	(4)
mean	235 - 175	249 -	204		253 -	224	
% change	- 26%	-	18%		-	12%	

[1] Longevity Research Institute (LRI, 1978).

[2] n = 884 (distribution was unavailable).

levels shows that the higher the level was initially, the greater the decrease. The mean percentage reduction in serum cholesterol level was greater (-26%) for the L.R.I. group than in either of the present study groups (PRIT group: -18%; AHA group: -12%).

Few variables were significantly different between diet groups. Within each group, however, there did appear to be improvement from month 0 to month 12 in many of the variables. Table 3.38 lists the variables for each group for which there was a significant difference between months 0 and 12. Student's t-test was used to determine these results.

The AHA group had strong significant changes ($p < 0.01$) in % fat intake, relative body weight and walking time, and moderate change ($p < 0.05$) in dietary fiber intake and serum HDL levels between months 0 and 12.

The PRIT group had strong significant changes ($p < 0.01$) in % fat intake, total fat intake, dietary cholesterol, dietary fiber, serum cholesterol, relative body weight, and walking time; and moderate change ($p < 0.05$) in carbohydrate intake and serum LDL levels between months 0 and 12.

TABLE 3.38 Parameters which showed a significant difference between months 0 and 12[1].

Parameter	AHA	PRIT
% fat intake	a[2]	a
fat intake total	-	a
carbohydrate intake	-	b
dietary cholesterol	-	a
dietary fiber	b	a
serum glucose (fasting)	-	b
serum cholesterol	-	a
serum HDL	b	-
serum LDL	-	b
RBW	a	a
walking time	a	a

[1]Determined by Student's t-test.

[2]Level of significance: a = $p < 0.01$ b = $p < 0.05$.

CHAPTER FOUR

DISCUSSION

A. Validity of Dietary Assessment

Determination of dietary intake is one of the most difficult tasks facing the nutritionist. Marr (1971) stated that there is no generally accepted method of measuring the dietary intake of free-living individuals. Although numerous methods have been devised, they are often suited only to specific situations. To evaluate dietary intake one must choose one's objectives; choose a method to meet those objectives; and respect the limitations imposed by that method (Beaton, 1973).

Objectives of Dietary Assessment for the Present Study:

i. to determine each subject's typical food intake prior to the study period.

ii. At specified intervals throughout the year, to determine:

a) whether each subject followed the prescribed meal plan.

b) the intakes of carbohydrate, protein, fat, fiber and cholesterol for each subject.

c) the motivation and subjective feelings of each subject.

After a review of dietary assessment, Mongeau (1973) concluded that a combination of methods was best. The following pair of methods was chosen for this study:

(1) a 48-hour recall
(2) a 3-day food record

The 48-hour recall was used to determine actual nutrient intake as closely as possible. It was used instead of the 24-hour recall because it is believed it improves the chance of obtaining a more representative intake because of the longer period recalled. Its advantages include:

a) its workability for the relatively small number of subjects

b) its accuracy for the nutrients on which this study concentrated

c) its standardized procedure for repeated interviews.

It has been stated that errors in estimating portion size are probably the largest source of error in dietary record-keeping (Young, 1952); however, once the study had begun the patients were required to weigh and measure food items and therefore were aware of the amounts they consumed. The use of food models for estimation of portion size further helped to minimize this error.

How often recalls should be repeated depends on the nature of the study. Balogh (1971) found that four recalls were necessary if the average caloric intake for one-half the sample was to be within $\pm$20% of the true mean; for other dietary components the number is even higher. The 48-hour recalls of this study were done nine times, at months 0,1,2,3,4,6,8,10 and 12. Average values of intakes from 1-6 months and 7-12 months were reported.

Monthly 3-consecutive-day food records, consisting of one weekend day and two weekdays, were assigned to determine the subjects' understanding of the exchange system and compliance with the diet.

Food records were completed mid-month and discussed at the subsequent visit to the Metabolic Center. These records provided a check on the recall estimations, but were not actually used for collecting data.

The education component was an essential part of the diet therapy. Witschl and co-workers (1978) found that enlisting the help of the family as a unit is an effective means of achieving dietary change. They also stated the need for follow-up in programs designed to lower cholesterol. Increases in cholesterol levels at three months after the termination of a cholesterol-lowering diet study indicated the importance of continuous monitoring, encouragement and education.

Weinsier (1974) described a 40-week education program for diabetic subjects. He reported a high level of adherence as shown by their laboratory results, a 99% attendance rate and a low dropout rate (4%). He attributed this success to:

a) teaching small groups
b) frequent follow-up
c) feedback on laboratory data
d) individualization of diet prescriptions
e) family involvement.

All these techniques were employed in the present study.

Heyden (1975) stated that "any diet modification de-emphasizing animal fat but emphasizing the use of fresh fruits, salads, vegetables, skim milk products and lean meat and fish will produce a general feeling of well-being -- the most powerful stimulant to follow diet modifications for extended periods of time. In another group of patients it is the close participation of the patient in the follow-up of his own laboratory studies."

The patients were all given extensive feedback on changes in weight, blood values and vascular parameters. A majority of the patients reported improved "well-being". These factors likely accounted for the low voluntary dropout rate of one out of 50.

B. Dietary Intakes

North Americans have been considered "typical" if they consume a diet high in fat and high in cholesterol (Connor & Connor, 1977). The approximate distribution of energy intake has been reported as 20% as protein, 40% as fat, and 40% as carbohydrate (Anderson, 1980). The two groups in the present study were "typical" upon entry into the study.

Dietary habits changed drastically during the study period. The AHA group consumed less fat and more carbohydrate to meet AHA specifications (Subcommittee on Diet and Hyperlipidemia, 1973). The Pritikin group consumed much less fat and much more carbohydrate. The total protein intakes were not significantly altered in either group and have not been emphasized in this investigation. The Pritikin group had a higher ratio of vegetable protein to total protein and appears to have a higher protein intake. Many of the subjects had difficulty consuming enough calories so foods such as breads, cereals and legumes were added throughout the study for extra calories. These increased the vegetable protein intakes as well as the carbohydrate intakes.

The Pritikin group consumed about one-half as much fat as the AHA group. The total fat intake of the Pritikin group at month 12 was significantly lower than at month 0 ($p < 0.01$); it was 28

and 23 g per day at month 12 for the males and females, respectively as compared to 91 and 43 g per day at month 0. There was a significant difference in the percentage of energy from fat at month 0 and 12 for both the AHA and Pritikin groups ($p < 0.01$). At month 0 fat intakes ranged from 34 to 42% of total calories; at month 12 fat intake was 29% of calories for the AHA group and about 15% of calories for the Pritikin group.

Cholesterol intakes were kept very low throughout the study. Again the PRIT group consumed about one-half as much as the AHA group. The PRIT group did not consume any visible sources of fat (i.e., butter, cream, sauces, etc.). Their only fat intake was from lean meat (a maximum of 3 oz. per day) and small amounts from grains and starches. Initially cholesterol intakes ranged from 166 to 330 mg per day. At month 12, the cholesterol intake of the Pritikin was significantly lower than at month 0; at month 12 it was 127 and 88 mg per day for the males and females, respectively.

The objective of 70% of energy from carbohydrate was not achieved by the PRIT group ($\bar{X} = 66\%$). The high fiber foods often caused abdominal discomfort and subjects occasionally did not consume their daily quota of grains, legumes and vegetables. However, the total carbohydrate intake of the Pritikin group was significantly higher at month 12 than at month 0 ($p < 0.05$); it increased from 210 to 269 g per day for the males and from 133 to 219 g per day for the females.

There was a significant difference between groups in dietary fiber intakes after dietary intervention. The Pritikin group's intakes of 45 g per day are seldom achieved in the population at

large. None the less, many patients consumed over 60 grams of dietary fiber per day (maximum of 83 g for one subject); showing a variability in tolerance of large fiber intakes. Only recently have food composition values for dietary fiber become available. Thus, a whole new area of investigation is now possible.

C. Lipid Response and Weight Loss

Plasma triglyceride levels appear to be more related to weight loss than are levels of other lipids. The males showed decreases in triglycerides of 21% and 29% for those members of either group with weight loss of $>$ 5.0 kg. Other investigators have found a similar response (Olefsky et al., 1974; Galbraith et al., 1966). Conversely, when subjects lost less than 5.0 kg, only small decreases or even slight increases in triglyceride levels resulted.

There was a lesser response in serum cholesterol with weight loss. HDL values increased substantially with weight loss and LDL values decreased with weight loss.

For both groups weight loss was significant ($p < 0.01$). Weight loss in 12 months was 4.1 kg for AHA group and 6.0 kg for the Pritikin group.

Relative body weight showed consistent positive correlations with triglycerides and cholesterol and consistent negative correlations with HDL. At month 12 the fasting blood sugar levels of the Pritikin group were significantly lower than they were at month 0 ($p < 0.05$).

D. Lipid Response

High fat and cholesterol intakes have been implicated as causing elevations of blood lipid levels (Connor, 1979). The implication is that lipid levels should fall when fat and cholesterol intakes are reduced. There were no strong correlations between fat intake and serum cholesterol levels, even though one group was consuming one-half as much fat as the other group. Fat intakes correlated negatively with walking times only at month 2. A direct effect of fat on the lipid responses obtained in this study is unlikely.

Correlations between individual fiber intakes and serum lipids were evident, and it appears that fiber is of dietary significance in this study. Carbohydrate intakes consistently showed a negative correlation with serum cholesterol ($p < 0.01$). We found correlations between total carbohydrate intake and serum cholesterol level at months 2,4,6 and 12, and between carbohydrate intake and walking time (month 4) and serum triglyceride levels (month 12). We also found significant correlations between dietary fiber intake and serum cholesterol levels at months 2,4 and 12 and between dietary fiber intake and walking time at month 4.

Kay and associates (1980) also found negative correlations ($p < 0.01$) between serum cholesterol and carbohydrate and dietary fiber intake from 24-hour recall data of 200 men.

Concern has developed over the source of fiber and whether certain types may have greater physiological effects. Wheat bran has shown inconsistent results in lowering serum cholesterol. No change occurred at 5 g/day(wheat bran as crude fiber), 50 g/day (All Bran), 17 g/day (hard wheat bran) or 26 g/day (soft wheat

bran) (Kay and Truswell, 1977_b, McDougall and Walker, 1978; Munoz et al., 1979). At higher doses of wheat bran, however, subjects experienced decreases in cholesterol of 10-25%. The Pritikin group consumed about 15 g wheat bran per day. Anderson recommended the use of oatmeal daily in his high fiber regimen. The addition of 12 g whole oats to the diet each day was associated with an 11% decrease in serum cholesterol. In the present study, hot oatmeal cereal was encouraged in both groups.

It has been suggested that various fiber components affect serum lipids and that more than one mechanism is involved. Some components (pectin, lignin) bind bile acids, other components interfere with absorption of lipids and carbohydrates, while other components increase the rate of passage of material through the intestine. The forms of fiber derived from fruits and vegetables such as pectin appear to be effective in reducing serum cholesterol when consumed in large quantities. Reductions in serum cholesterol of 4 to 12% have been reported (Kay, 1977; Stasse-Wolthuis, 1980; Miettienen1977; Kies, 1977).

Pectin fiber, found in fruit, has been shown to decrease serum cholesterol by 13% (at intakes of 15 g/day), 13% (25 g/day) and 13 mg (28 g/day) (Kay and Truswell, 1977_a, Anderson and Chen, 1979; Stasse-Wolthuis et al., 1980). In the present study, both groups increased their fruit intake.

Anderson (1980) concluded that a diet high in fiber from various fiber sources results in lipid-lowering. In his study, fiber sources included grains (40% of total fiber intake), fruits (9%) and vegetables (51%). Serum cholesterol levels decreased.

In the present study, the per cent contribution of different fiber sources (AHA ; PRIT group respectively) was as follows: cereals 50 ; 56%, vegetables 28 ; 25%, and fruit 20 ; 19%. However, the Pritikin group's intake of fiber was about twice that of the AHA group.For the Pritikin group, intakes averaged 27 to 30 g of dietary fiber per 1000 kilocalories; compared to 15 to 20 g per 1000 kcal for the AHA group.

Serum cholesterol was negatively correlated with dietary fiber.Significant decreases in serum cholesterol levels ($p < 0.01$) and serum LDL levels ($p < 0.05$) were obtained with the Pritikin diet at month 12. No significant change occurred in HDL levels. The LDL/HDL ratios were lower at month 12 (-12%). The decrease in total cholesterol accompanied by no change in HDL cholesterol could indicate an increased transport of cholesterol as HDL in these patients.

For the AHA group, the only significant change at 12 months was an increase in serum HDL levels ($p < 0.05$). There was a greater reduction in serum cholesterol levels in the first six months of the study than in the last six months. The reduction in serum cholesterol levels was less in the AHA group than in the Pritikin group. The increase in HDL cholesterol levels was surprising. Schaefer and co-workers (1981) found that HDL levels decreased with an AHA diet and that there was no change in LDL/HDL ratio. Carlson and co-workers (1977) investigated patients with high triglyceride levels. After treatment, the decrease in triglyceride levels was accompanied by increases in LDL and HDL levels. Minimal change occurred in the LDL/HDL ratio. In the present study, many patients

had high serum triglyceride levels; the increase in HDL levels may be associated with a decrease in triglycerides. The LDL/HDL ratios were lower at month 12 (-10%) as was the case with the Pritikin group. Improved ratios were also reported elsewhere with the use of a fat-modified diet supplemented with fruit, vegetable and cereal fiber (Lewis et al., 1981).

In the present study, little change in total triglyceride levels was evident. However, this does not rule out the possibility that changes in composition or concentration of individual triglyceride transporting lipoproteins did occur.

Not only dietary parameters, but a number of other parameters, such as cigarette smoking, have additional small effects on lipid response.

E. Improvement in Health as Measured by Improvement in Walking Ability

The majority of the patients in both groups improved their walking times and distances. The greater improvement in walking ability was evident in the Pritikin group. The improvement in the length of time the patients could walk on a treadmill before experiencing claudication was significantly greater for the Pritikin group males than for the AHA group males at six months ($p < 0.05$). Unfortunately the walking times are of limited value. The speed on the treadmill was increased every ten minutes; it was 1.50 mph for the first ten minutes, 2.25 mph for the second ten minutes, and 3.00 mph for the next ten minutes. Thirty minutes was the maximum time allotted patients on the treadmill.

Walking times were converted to walking distances (1811 meters is maximum). The improvement in walking distance at six months was 636 meters for the AHA group and 797 meters for the Pritikin group. Even at six months, there were some patients whose condition improved to the extent that they could walk the maximum of 1811 meters. The number of patients who could walk 1811 meters was greater for the Pritikin group than the AHA group. At six months 6 out of 22 (27%) of the Pritikin patients and 1 out of 17 (6%) of the AHA patients could walk 1811 meters. At 12 months 11 out of 22 (50%) of the Pritikin patients and 1 out of 16 (6%) of the AHA patients could walk 1811 meters. The Pritikin group appeared to respond better to diet therapy. Although no dietary components showed strong correlations with walking time, the Pritikin group walked farther.

In the present study, where type II and Type IV HLP were normalized, the LDL/HDL values were less than average at month 12. Also, patients who showed the greatest improvement in walking had the lowest LDL/HDL ratios. Nine patients decreased their LDL/HDL ratios by ≥ 1.0. These individual cases are presented in Appendix E_3. Five of these were from the AHA group (3M, 2F) and four were from the Pritikin group (4M). These subjects had in common:

1. weight loss (9/9)
2. decreased triglycerides (8/9)
3. decreased cholesterol (8/9)
4. increased HDL (6/9)
5. decreased LDL (9/9)
6. decreased VLDL (8/9)
7. large increases in W.D. (8/9)

Perhaps some dietary components affect walking times. However, evidence of such did not appear in this pilot study. Single dietary factors must be studied in controlled clinical trials. What this study did show was that P.V.D. patients experienced weight loss and functional improvement on a diet and exercise program.

Our findings concur with McAllister (1976) who investigated the progress of patients with intermittent claudication managed without surgery. These patients were instructed to increase total distance walked from day to day; to follow a low-calorie diet (if overweight) or a low fat, low cholesterol diet (if lipids elevated), or a strict diabetic diet (if diabetic); and to quit smoking. The author conducted a 6-year follow-up and found that the patient with intermittent claudication without associated grave signs on this program has a greater than 50% chance of improving and a greater than 60% chance that his disease will not progress during a 5-6 year period.

SUMMARY

Walking time and distance improved in almost all of the patients. Generally there appeared to be decreases in triglycerides, cholesterol and LDL and increases in HDL in almost all of the patients. Significant weight loss occurred in both groups. Many previously poor dietary habits improved, and many patients remarked that they experienced a greater sense of well-being. For most

parameters neither diet showed significantly better results than the other, the fact remains that a diet and exercise program helped these patients both physically and emotionally. Also, the fact that the patients successfully followed the program for an entire year with little deviation is truly gratifying. Lampman (1977) stated that patients need to participate regularly in formal programs in order to maintain adherence to diet and exercise regimes.

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APPENDIX A_1

The usual method of assessing progression or regression of atherosclerosis is by angiography, an inconvenient, expensive and painful procedure not without risk to the patient. Non-invasive procedures using an analysis of Doppler flow pulse waves, measurement of segmental systolic pressures by Doppler flowmeter, and measurement of ankle systolic pressure response to exercise, provide sensitive information about the severity and direction of change of the disease. [1,2,3,4,5,6,7,8,9]

The most common functional assessment of peripheral vascular disease is through measurement of walking time or walking distance on a treadmill. While a useful estimate of the degree of disability experienced by the patient, the test has been shown not to correlate highly with the more objective measurements. Observed improvement in walking time or distance in several studies has therefore been variously attributed to improved peripheral utilization of oxygen, improved glycolytic and oxidative metabolic capacity and improved walking technique rather than to actual vascular changes. [10]

APPENDIX A_1 - REFERENCES

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APPENDIX B_1

UNIVERSITY OF ALBERTA HOSPITAL

CONSENT TO PARTICIPATE IN A STUDY

PATIENT_____ DATE _____ TIME _____

1. I agree to participate in an investigation and in relation to this hereby authorize Dr. _____ and/or such assistants as may be selected by him, to perform the following procedure(s):

_____

_____

_____

2. Dr. _____ has explained the purpose of this study and I understand this, the risks involved and the nature of the procedure(s) outlined in Paragraph 1. (Where pertinent, a typed sheet detailing this should be prepared by the Investigator and attached to this form).

3. I acknowledge that no guarantees have been made to me as to the results of the treatment.

_____ _____

Witness Signature of Patient

_____

If the patient is unable to sign or is under 18 years of age, complete the following:

The patient is a minor (_____ years of age).

OR

The patient is unable to sign because _____.

As the closest relative or legal guardian I hereby sign on his/her behalf:

_____ _____ _____

Witness Signature Relationship

APPENDIX B_2

PERIPHERAL VASCULAR DISEASE STUDY

Assessment of arterial disease in this study requires a number of non-invasive physiological measurements. Blood pressure will be measured using an ultrasonic flowmeter before and after exercise on a treadmill. The exercise period will be limited by pain in the legs or by the achievement of submaximal heart rates. The risks of heart problems associated with this level of exercise are rare. However, if they should occur, treatment is immediately available.

Blood flow waveforms will be obtained using ultrasound. Risks associated with this non-invasive measurement are negligible.

I consent voluntarily to these tests which have been fully explained to me.

SIGNED _____

WITNESS _____

DATE _____

APPENDIX B_3.

I, the undersigned _____ hereby request Dr. Ken Hutchison and Dr. Gordon Brown and their associates to arrange a trial for me involving careful dietary treatment, which has been recommended in controlling blood vessel disease in the limbs.

I agree to participate in the study to determine the effect of one or other of two special diets, each of which has been designed, but not proven, to lessen the effects of blood vessel disease.

I understand that the diet selected for me may or may not be helpful. I understand that there is no conclusive evidence that either diet is harmful for a patient under careful observation during the trial period. I understand that some laboratory tests will be required and I understand that not all of these will be necessary for the care of my condition. The following tests will be determined monthly from blood specimens: glucose, cholesterol, and triglycerides. The following blood tests will be determined initially, at one month, then three, six, and twelve month intervals: lipoproteins, TSH, platelet adhesion and aggregation.

I understand that withdrawal from this study will be permitted at any time without malice or interference by the physicians and their assistants. I understand that a reasonable amount of time will be required to assess whether or not one or other of the diets is effective.

DATE _____ _____
(Patient's signature)

(Witness) _____

APPENDIX C_1

DIET INFORMATION

1) DO YOU HAVE ANY FOOD ALLERGIES?

2) HAVE YOU EVER FOLLOWED A DIET BEFORE? YES_____ NO _____

IF YES - WHAT TYPE? _____

HOW LONG? _____

WHAT RESULTS? _____

_____

_____

DID YOU WEIGH AND MEASURE YOUR FOOD? _____

3) WHO DOES THE COOKING AT HOME? _____

FOR HOW MANY? _____

WOULD YOU/THEY BE WILLING TO TRY NEW RECIPES? _____

WOULD YOU/THEY BE WILLING TO BUY SOME SPECIAL PRODUCTS?
(i.e., Sucaryl, Pam, fat-free products) _____

4) DOES YOUR JOB REQUIRE THAT YOU EAT AWAY FROM HOME? YES _____

NO _____

IF YES, NO. OF MEALS PER WEEK _____

EATING FACILITY - CAFETERIA _____

RESTAURANT _____

BAG LUNCH _____

OTHER _____

HOW OFTEN WOULD YOU EAT OUT SOCIALLY?

MEALS PER WEEK _____

HOUSE _____

RESTAURANT _____

5) OTHER COMMENTS:

MOTIVATION:

SPOUSE'S ATTITUDE:

APPENDIX C_2

48 HOUR DIETARY RECALL

NAME: _____

CHART #: _____

TIME OF DAY Enter Time from 00:01 to 24:00 In hours and minutes	PLACE OF EATING 1. Home 2. School 3. Work, Field 4. Restaurant 5. Other SOURCE: 1. Home 2. Self Purchased 3. Other FOOD CODE:	FOOD OR DRINK	WORK AREA	AMOUNT CONSUMED Whole Decimal	FOOD UNIT or MODEL NO.

APPENDIX C_3

I. Interviewing

Efficiency and accuracy are the goals but there is no single way to conduct an ideal interview. The following suggestions are made to assist each interviewer in developing his own communication skills.

Verbal and Non-Verbal Communication

The non-verbal can be as important as the verbal part of the interview process. An unconscious nod of the head or grimace will encourage or discourage further responses by the person being interviewed. The interviewer should attempt to eliminate any action which will unconsciously influence the person being interviewed. Inappropriate or distracting wearing apparel will have a similar effect upon the interviewing process.

The interviewer must be sincere and straight-forward in the verbal part of the interview. After several interviews there is a temptation for the interviewer to use a monotone and become machine-like in his responses to the person being interviewed. Keep in mind that while it may be your last interview for the day it is the other person's first with you.

Forms

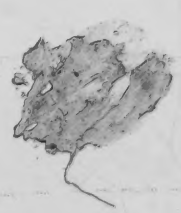
As you get used to using forms you will be tempted to take short cuts in instructions. Misunderstanding on the part of the individual being interviewed will result in less accuracy in his responses so be sure to give full instructions to each person being interviewed.

Occasionally a person will ask to see what you have written down, or previous to an interview, the form which you will use. He will probably feel more comfortable and therefore respond more accurately after you have shown him the form.

The Clinical and Dietary Interview

The dietary and clinical interviewers have to be completely at ease with the forms and the food models so that they assist the communication process. A fumbled model or a lost place on a form distracts the individuals attention and leads to less accurate responses.

1. When not in use food models should be off the table and out of sight to the person being interviewed.



2. All models in a set should be placed on the table at the same time.

3. Models should all be equidistant from the person being interviewed.

4. Models should be presented in graduated rather than random order.

RECORDING THE 24-HOUR FOOD INTAKE

I. How to take a 24-hour recall

Use a friendly approach when meeting your participant as they will be apprehensive; continue this rapport with small talk, keeping the respondant aware of his importance. Give a very concise explanation of what will be expected during the dietary interview. "We are interested to know what you ate yesterday. Will you please tell me no matter if it is just a bite, a cup of coffee, a coke, candy or a beer?"

"Let's start with yesterday morning when you got up. What was the first thing that was eaten or drunk besides water?" "What other foods were eaten at this time? What was the next time you had something to eat or drink?" Continue through the day until the 24-hour period is covered.

Assist the respondant by helping him recall his activities yesterday. When this list of foods has been completed go back to the first food mentioned and question the respondant about the amount of food consumed, using the appropriate models. "Do you see any model here that is about the size of what you had?" Allow the respondant to speak of parts or multiples of a model. In some cases, the respondant may offer information regarding the amount eaten as he recalls his food intake -- record these when first mentioned and clarify later.

eg.: Now we'd like to know how much of these foods you've had. You said you had reconstituted orange juice for breakfast. How much did you have? Do any of these glasses help you tell me how much? How filled was your glass? Continue for every food using appropriate models.

II. Models for assessing portion size

The use of food models in surveys is not new. However, the models to be used in Nutrition Canada are unique in that they had been designated in conjunction with a program for data processing. Nutritionists and dietitians are familiar with more conventional survey methods which consist of:

a) collecting dietary information and recording it in household portions, then,

b) converting the household portion to gram weights so that

c) nutrient values may be determined.

The conversion intake to gram weights is necessary because most tables of nutrient values are based on gram weight of foods.

For Nutrition Canada, an electronic computer will be used for determining the nutrient values of food consumed. The program for processing the data will be based on a food composition table which includes the 2483 food items appearing in U.S. Department of Agriculture Handbook No. 8 (1963), plus additional codes needed for food unique to Canada and for other foods respondents report they consume. The master food table lists 17 constituents based on the amount of each nutrient in 100 grams of the specific food item.

The models have been designed to assist interviewers in securing from the respondent the amounts of food consumed. They have also been designed so that the computer can take the alphabetic code assigned to each model, make necessary mathematical computations and arrive at (1) the grams of a food consumed then (2) the nutrients from the amount of food consumed. An over-simplified explanation of what will occur is demonstrated by the following example:

1. A respondent may answer: "I ate lettuce in the amount of twice model S yesterday."

   a) The interviewer will record as follows:

Food Item	Amount Consumed	Food Model No.
Lettuce	2	S

   b) The computer will automatically know the gram weight of lettuce based on the food code numbers recorded in the Food Code Columns, understand that model "S" refers to 3/4 cup, and make necessary calculations.

2. The respondent may have answered: "I ate spaghetti and meat sauce in the amount of twice model S yesterday."

   a) The interviewer will record as follows:

Food Item	Amount Consumed	Food Model No.
Spaghetti and meat sauce	2	S

b) The computer's automatic calculations know that the gram weight and nutrient values for spaghetti and meat sauce are different from lettuce since the food code is different. It will then make necessary calculations to get nutrient values for this different item although measured with same model.

APPENDIX C_4

II. Description of Food Models

The food models must be presented in a way that does not bias the respondent. All of one type must be presented simultaneously.

Models	Code	Full volume (oz)
Cups	A	6
	S	10
Glasses	B	11
	V	8
	Q	3.75

Possible usage of glasses and cups: any liquid you normally measure in cups, i.e., juice, beverage, soups.

Spoons	SS	.75
	CC	.5
	M	.36
	E	.22

Possible usage of spoons: any food item you normally measure in tablespoons or teaspoons, i.e., sugar, baby food, jams, peanut butter, oils, salad dressings, mayonnaise, whipped cream.

Bowls	A	10
	D	4

Possible usage of bowls: any food which can be measured in these dishes, i.e., custard, pudding, canned or stewed fruit, raw berries, ice cream, dry or cooked cereals, soups, stews.

Mounds	C	16
	A	10
	S	6
	Z	2
Cylinder	D	4

Possible usage of mounds and cylinder: any non-liquid food you normally serve on a plate and measure in cups such as mashed potatoes, cooked vegetables, green salads, pasta casseroles. The cylinder can be used for foods such as broccoli, green and yellow beans, cooked whole carrots, asparagus.

Butter Pat	E	.2
French Bread Model	UNIT	
Pie Model	D	4

Models	Code	Full volume (oz)
	Q	5
Discs, Squares,	E	.22 --- 2.6 (varies
Retail Cuts	M	.36 --- 4.3 with thick-
	CC	.5 --- 6.0 ness)
	MM	.75 --- 9.0
	G	1.0 --- 12.0
	H	1.25 --- 15.0
	R	2.5 --- 30.0

Possible usage of discs, squares or retail cuts: cooked meats, roast, steak, cold cuts, luncheon meats. The discs and squares may serve as a guide for the size of pancakes or foods of similar shape.

Meal & Fish	H	1.25
Models	D	4.00

Possible usage of meat models: beef, veal, lamb as stew, pork, etc.

Boxes	P	122 (gm)
	L	95
	X	38

Possible usage for boxes: cake, jellyrolls, breads: gingerbread, corn, banana.

Balls	SM	.75 (oz)
	MD	2.5
	LG	4.0
	XLG	6.0

Possible usage for balls: meatballs, round type baked goods like popovers.

APPENDIX D_1

LIFESTYLE QUESTIONNAIRE

SMOKING STATUS

NEVER _____

EX-SMOKER _____ (NO. OF YEARS SMOKING/NON-SMOKING)

SMOKER _____ SINCE WHEN? _____

NO.OF CIGARETTES PER DAY? _____

OTHER (CIGARS?, PIPES?) _____

ALCOHOL STATUS

NEVER _____

OCCASIONALLY _____

FREQUENTLY _____ NO. OF DRINKS PER WEEK _____

TYPES OF ALCOHOL _____

EXERCISE STATUS (DISTANCE & TIME)

DAILY: _____

_____

WEEKLY: _____

_____

WOULD YOU BE WILLING TO BUY SPECIAL ITEMS FOR THE DIET? _____

(i.e., scales, measuring cups)

WILL YOU BE AVAILABLE FOR FOLLOW-UP VISITS? YES _____ NO _____

(i.e., once/month for 12 months)

APPENDIX D_2

Suggested Ideal Weights According to Stature for Adults

MEN over 25 years of age

HEIGHT centimeters	SMALL FRAME (less than 15.2 cm wrist) kg	MEDIUM FRAME (15.2 to 17.8 cm wrist) kg	LARGE FRAME (more than 17.8 cm wrist) kg
154	51 - 54	54 - 59	57 - 64
156	52 - 56	55 - 60	59 - 65
159	54 - 57	56 - 62	60 - 67
162	55 - 59	58 - 63	61 - 69
164	56 - 60	59 - 65	64 - 71
167	58 - 62	62 - 67	64 - 73
169	60 - 64	63 - 69	67 - 75
172	62 - 66	64 - 71	69 - 77
174	64 - 68	66 - 73	70 - 79
176	66 - 70	68 - 75	72 - 81
178	67 - 72	70 - 77	74 - 83
181	69 - 74	72 - 79	76 - 86
184	71 - 76	74 - 82	79 - 88
186	73 - 78	76 - 84	81 - 90
189	74 - 79	78 - 86	83 - 93

WOMEN over 25 years of age

HEIGHT centimeters	SMALL FRAME (less than 15.2 cm wrist) kg	MEDIUM FRAME (15.2 to 16.5 cm wrist) kg	LARGE FRAME (more than 16.5 cm wrist) kg
142	42 - 44	44 - 47	47 - 54
144	43 - 46	44 - 50	48 - 55
147	44 - 47	46 - 51	49 - 57
149	45 - 48	47 - 53	51 - 58
152	46 - 50	49 - 54	52 - 60
155	48 - 51	50 - 56	54 - 62
157	49 - 53	51 - 57	55 - 64
160	50 - 54	53 - 60	57 - 66
162	52 - 56	54 - 61	59 - 66
165	54 - 58	56 - 63	60 - 68
167	55 - 59	56 - 65	62 - 70
170	57 - 61	60 - 67	64 - 72
172	60 - 64	62 - 69	66 - 74
174	62 - 65	64 - 70	68 - 76
178	64 - 67	65 - 72	69 - 79

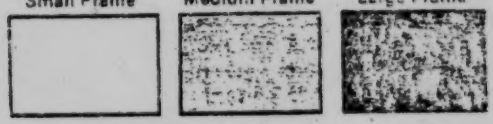
Adapted from Metropolitan Life Insurance Company

Note: Weights are given for an individual wearing street clothes (no shoes).

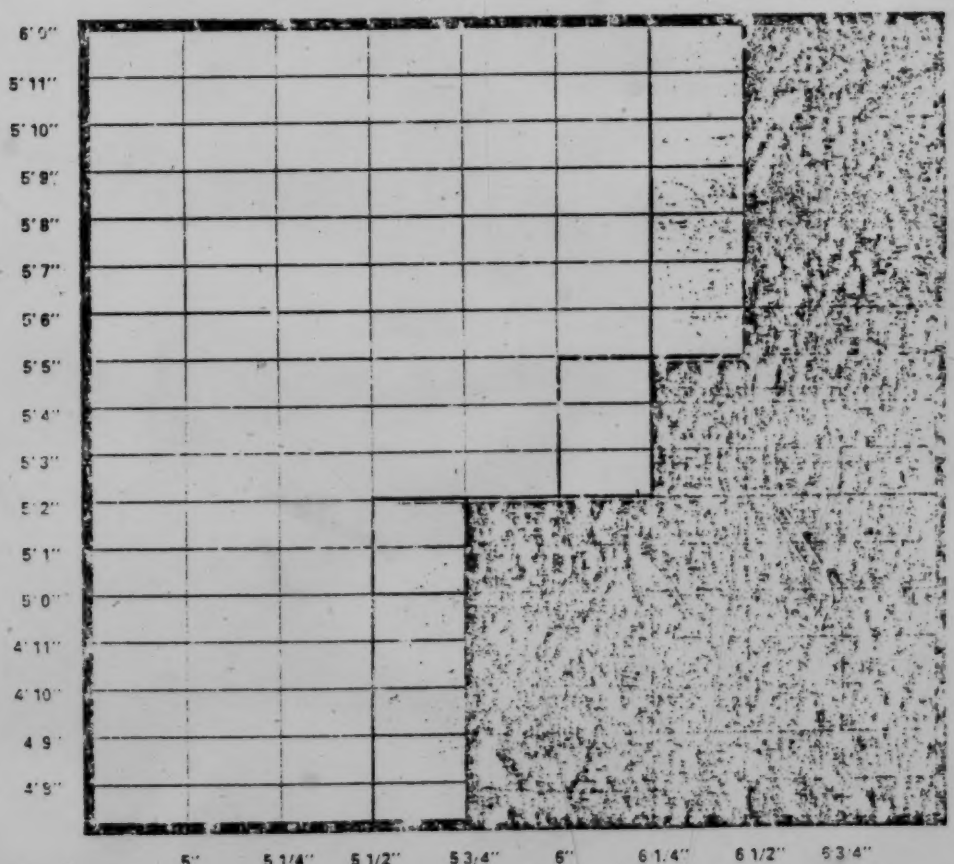
APPENDIX D3

Body Frame Type

Small Frame | Medium Frame | Large Frame



The wrist is measured distal to styloid process of radius and ulna at smallest circumference. Use height without shoes and inches for wrist size to determine frame type from this chart.



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APPENDIX D_4

WOMEN % FAT

TOTAL SKINFOLD (mm)	FAT	TOTAL SKINFOLD (mm)	FAT	TOTAL SKINFOLD (mm)	FAT	TOTAL SKINFOLD (mm)	FAT
20	15.5	40	24.5	60	30.0	80	34.0
21	16.1	41	24.8	61	30.2	81	34.2
22	16.7	42	25.1	62	30.4	82	34.4
23 (.06)	17.3	43 (.03)	25.4	63 (.02)	30.6	83 (.02)	34.6
24	17.9	44	25.7	64	30.8	84	34.8
25	18.5	45	26.0	65	31.0	85	35.0
26	19.0	46	26.3	66	31.3	86	35.2
27 (.05)	19.5	47 (.03)	26.6	67 (.03)	31.6	87 (.02)	35.4
28	20.0	48	26.9	68	31.9	88	35.6
29	20.5	49	27.2	69	32.2	89	35.8
30	21.0	50	27.5	70	32.5	90	36.0
31	21.4	51	27.8	71	32.7	91	36.1
32	21.8	52	28.1	72	32.9	92	36.2
33 (.04)	22.2	53 (.03)	28.4	73 (.02)	33.1	93 (.01)	36.3
34	22.6	54	28.7	74	33.3	94	36.4
35	23.0	55	29.0	75	33.5		
36	23.3	56	29.2	76	33.6		
37 (.03)	23.6	57 (.02)	29.4	77 (.01)	33.7		
38	23.9	58	29.6	78	33.8		
39	24.2	59	29.8	79	33.9		

APPENDIX D_4

MEN % FAT

TOTAL SKINFOLD (mm)	FAT	TOTAL SKINFOLD (mm)	FAT	TOTAL SKINFOLD (mm)	FAT	TOTAL SKINFOLD (mm)	FAT
15	5.50	36	15.80	56	21.20	76	25.20
16	6.20	37	16.10	57	21.40	77	25.40
17 (.07)	6.90	38 (.03)	16.40	58 (.02)	21.60	78	25.60
18	7.60	39	16.70	59	21.80	79 (.02)	25.80
19	8.80	40	17.00	60	22.00	80	26.00
20	9.00						
21	9.50	41	17.30	61	22.20	81	26.10
22	10.00	42	17.60	62	22.40	82	26.20
23 (.05)	10.50	43 (.03)	17.90	63 (.02)	22.60	83 (.01)	26.30
24	11.00	44	18.20	64	22.80	84	26.40
25	11.50	45	18.50	65	23.00	85	26.50
26	11.90	46	18.80	66	23.20	86	26.70
27	12.30	47	19.10	67	23.40	87	26.90
28 (.04)	12.70	48 (.03)	19.40	68 (.02)	23.60	88 (.02)	27.10
29	13.10	49	19.70	69	23.80	89	27.30
30	13.50	50	20.00	70	24.00	90	27.50
31	13.90	51	20.20	71	24.20		
32	14.30	52	20.40	72	24.40		
33 (.04)	14.70	53 (.02)	20.60	73 (.02)	24.60		
34	15.10	54	20.80	74	24.80		
35	15.50	55	21.00	75	25.00		

APPENDIX D_5 DATE _____

PATIENT INFORMATION - ANTHROPOMETRIC MEASURES

NAME: _____ ADDRESS _____

BIRTHDATE: _____ _____

MARITAL STATUS: _____ PHONE (HOME): _____

OCCUPATION: _____ (BUSINESS): _____

AHC #: _____

MEDICAL HISTORY

HEIGHT: _____ DOCTOR: _____

WEIGHT: _____

DO YOU FEEL YOU ARE UNDER/OVER WEIGHT? _____

IF SO, HOW MUCH? _____

IDEAL BODY WEIGHT _____

HISTORY:

	MONTHS	
SKINFOLDS:	0	12
BICEPS (mm)		
TRICEPS (mm)		
SUB-SCAPULA (mm)		
ILIAC CREST (mm)		
TOTAL		
% BODY FAT		
MID-ARM CIRCUMFERENCE		
WRIST CIRCUMFERENCE		
FRAME SIZE		

APPENDIX E_1

SUGAR AND STARCH INTAKES

GROUP	(n)	SUGAR (g/day)	STARCH (g/day)	SUGAR:STARCH RATIO
			Mean Values	
Month 0				
AHA				
males	(16)	90	102	0.9
females	(7)	74	76	1.0
PRIT				
males	(21)	91	109	0.8
females	(6)	64	51	1.3
Month 6				
AHA				
males	(16)	72	90	0.8
	(6)	60	71	0.9
PRIT				
males	(21)	85	157	0.5
females	(6)	66	115	0.6
Month 12				
AHA				
males	(15)	86	112	0.8
females	(5)	65	85	0.8
PRIT				
males	(18)	77	140	0.6
females	(6)	86	130	0.7

APPENDIX E_2

Changes in walking distance after one year and LDL/HDL ratio at month 12.

AHA					PRIT				
		WD Change (m)	LDL/HDL (mo.12)				WD Change (m)	LDL/HDL (mo.12)	
Subject No.	sex				Subject No.	sex			
08	F	+1435	1.9	(-1.5)*	06	F	+1730	2.8	(+.1)
05	M	+1122	2.0	(-.2)	46	M	+1440	3.1	(-.8)
53	F	+999	2.3	(-2.0)	02	M	+1297	2.8	(0)
29	F	+945	3.5	(-.8)	25	F	+1263	2.0	(-.1)
09	M	+868	2.5	(-.3)	07	M	+1156	3.8	(-.3)
42	M	+817	2.8	(-1.2)	16	M	+1139	1.2	(-2.0)
13	M	+734	1.8	(-.3)	54	M	+1060	2.4	(-.7)
32	M	+578	2.3	(-1.5)	24	F	+969	3.0	(+.2)
14	M	+560	1.6	(+.5)	51	M	+963	3.9	(-1.0)
49	M	+543	4.8	-	18	M	+956	4.0	(-.6)
35	M	+409	3.1	(+.1)	39	M	+757	2.9	(-.8)
11	M	+322	3.5	(-.6)	52	M	+736	2.6	(+.1)
04	M	+253	2.7	(+.1)	33	M	+725	2.1	(-.7)
37	F	+76	4.2	(+.9)	43	M	+718	3.9	(+.5)
38	M	-56	1.8	(-1.2)	48	M	+707	4.2	(-.5)
50	F	-185	3.6	(+.6)	17	M	+493	2.2	(-.4)
					36	M	+239	4.4	(+.8)
					22	M	+195	2.4	(-1.4)
					15	M	+181	3.4	(-1.1)
					28	M	+181	3.2	(+.2)
					20	F	-241	2.0	(-.6)
					26	M	-332	3.9	(-.3)

* change from month 0.

APPENDIX E_3

Subjects who showed decreases in the LDL/HDL ratio ≥ 1.0 after one year.

Subject no.	sex	group		Wt. (kg)	WD (m)	TG (mg)	CHOL (mg)	HDL (mg)	LDL (mg)	VLDL (mg)
Case no. 1			mo. 0	62.2		174	264	52	177	35
08	F	AHA	mo.12	50.3		94	205	65	121	19
				-11.9	+1435	-80	-59	+13	-56	-16
Case no. 2										
53	F	AHA	mo. 0	84.0		158	261	43	186	32
			mo.12	71.4		334	292	69	156	67
				-12.6	+999	+176	+31	+26	-30	+35
Case no. 3			mo. 0	78.0		162	246	43	171	32
42	M	AHA	mo.12	75.7		139	190	43	119	28
				-2.3	+817	-23	-56	-	-52	-4
Case no. 4			mo. 0	97.0		176	228	40	153	35
32	M	AHA	mo.12	92.7		130	189	50	113	26
				-4.3	+578	-46	-39	+10	-40	-9
Case no. 5			mo. 0	71.0		100	197	44	133	20
38	M	AHA	mo.12	63.2		96	181	58	104	19
				-7.8	-56	-4	-16	+14	-29	-1
Case no. 6			mo. 0	77.7		184	229	46	146	37
36	M	PRIT	mo.12	68.2		106	167	67	79	21
				-9.5	+1139	-78	-62	+21	-67	-16
Case no. 7			mo. 0	93.6		161	296	45	219	32
51	M	PRIT	mo.12	74.4		78	221	42	163	16
				-19.2	+963	-83	-75	-3	-56	-16
Case no. 8			mo. 0	65.0		191	219	38	143	38
21	M	PRIT	mo.12	64.3		126	184	47	112	25
				-.7	+195	-65	-35	+9	-31	-13
Case no. 9			mo. 0	85.0		215	248	37	168	43
15	M	PRIT	mo.12	73.0		200	198	36	122	40
				-12.0	+181	-15	-50	-1	-46	-3

APPENDIX E_4

Serum cholesterol values - PRIT Group

Subject No.	Sex	Months 0	1	2	4	6	12
01	F	253	225	237	247	221	-
06	F	252	182	206	222	204	235
20	F	218	189	197	193	196	181
24	F	175	181	202	220	203	209
25	F	211	188	198	166	198	183
45	F	409	311	341	282	403	-
02	M	150	137	142	133	139	142
07	M	274	205	214	228	241	239
15	M	248	170	194	190	202	198
16	M	229	191	223	213	207	167
17	M	272	181	194	192	198	247
18	M	275	262	245	232	296	291
21	M	219	206	197	179	-	-
22	M	219	174	171	167	195	184
26	M	243	193	171	212	235	237
28	M	241	203	196	225	213	236
33	M	255	206	238	236	231	229
36	M	212	193	167	211	196	222
39	M	255	216	216	202	211	221
40	M	250	188	199	217	-	-
43	M	290	219	204	202	230	247
44	M	250	-	245	230	-	-
46	M	273	234	232	278	320	300
48	M	287	239	236	250	274	270
51	M	296	163	196	175	182	221
52	M	219	243	-	203	249	256
54	M	245	196	193	195	210	208

APPENDIX E_4

Serum triglyceride values - PRIT Group

Subject No.	Sex	Months 0	1	2	4	6	12
01	F	110	111	117	105	87	-
06	F	140	126	95	137	145	96
20	F	270	195	186	186	164	154
24	F	113	131	112	141	149	143
25	F	83	136	106	69	89	53
45	F	1630	447	798	525	1650	-
02	M	170	210	154	159	122	169
07	M	168	100	112	147	129	147
15	M	215	110	157	139	329	200
16	M	184	120	166	152	132	106
17	M	157	105	136	165	166	169
18	M	147	121	188	224	205	271
21	M	141	143	133	126	-	-
22	M	191	153	195	140	185	126
26	M	258	179	171	161	180	186
28	M	144	131	112	164	141	112
33	M	89	76	99	91	97	110
36	M	249	107	123	214	266	269
39	M	235	184	158	168	136	134
40	M	161	125	147	174	-	-
43	M	302	202	196	234	238	176
44	M	200	-	182	143	-	-
46	M	190	240	183	289	350	321
48	M	240	213	178	169	178	160
51	M	161	66	74	78	101	78
52	M	147	156	-	65	88	166
54	M	280	166	199	157	162	101

APPENDIX E_4

Serum HDL values - PRIT Group

Subject No.	Sex	Months 0	1	2	4	6	12
01	F	70	70	73	77	85	-
06	F	60	54	49	56	57	65
20	F	45	46	48	-	50	50
24	F	40	40	45	43	51	45
25	F	63	38	53	52	50	57
45	F	-	37	42	37	57	-
02	M	30	31	44	36	30	30
07	M	47	45	47	47	44	44
15	M	37	35	37	42	35	36
16	M	46	40	60	47	50	67
17	M	67	58	55	53	60	66
18	M	44	54	44	37	47	47
21	M	-	65	78	62	-	-
22	M	38	35	42	37	46	47
26	M	37	41	34	40	40	41
28	M	53	57	41	45	40	51
33	M	63	67	70	74	64	66
36	M	35	41	35	31	30	31
39	M	44	42	46	41	44	50
40	M	56	43	51	59	-	-
43	M	52	45	38	40	43	43
44	M	40	-	44	45	-	-
46	M	48	42	42	47	59	57
48	M	42	44	40	44	48	46
51	M	45	37	37	37	36	42
52	M	55	54	-	56	59	62
54	M	46	48	39	43	51	55

APPENDIX E_4

Serum LDL values - PRIT Group

Subject No.	Sex	Months 0	1	2	4	6	12
01	F	161	133	141	149	92	-
06	F	164	103	138	139	118	151
20	F	119	104	112	-	113	100
24	F	112	115	135	149	122	135
25	F	131	123	124	100	130	115
45	F	-	185	139	140	16	-
02	M	86	64	67	65	85	84
07	M	193	140	145	152	171	166
15	M	168	113	126	120	101	122
16	M	146	127	130	136	131	79
17	M	174	102	112	106	105	147
18	M	202	184	163	150	208	190
21	M	-	112	92	92	-	-
22	M	143	108	90	102	112	112
26	M	154	116	103	140	159	159
28	M	159	120	133	147	145	163
33	M	174	124	148	144	148	141
36	M	127	131	107	137	113	137
39	M	164	137	138	128	140	144
40	M	162	120	119	123	-	-
43	M	178	134	127	115	139	169
44	M	170	-	165	156	-	-
46	M	187	144	153	173	191	179
48	M	197	152	160	172	190	192
51	M	219	113	144	122	126	163
52	M	135	158	-	134	172	161
54	M	143	115	114	121	127	133

APPENDIX E_4

Serum cholesterol values - AHA Group

Subject No.	Sex	0	1	2	4	6	12
08	F	264	206	185	209	203	205
10	F	272	269	269	280	232	-
29	F	252	241	304	342	272	293
37	F	301	280	308	343	355	327
47	F	349	303	316	325	258	-
50	F	228	-	194	202	202	221
53	F	261	243	239	247	-	292
04	M	209	176	179	192	171	194
05	M	248	208	203	186	171	207
09	M	203	194	185	200	178	188
11	M	269	214	218	205	243	225
13	M	210	190	148	182	175	183
14	M	132	215	192	202	193	190
19	M	348	292	256	312	285	316
23	M	195	187	176	175	172	225
32	M	228	166	184	207	184	189
34	M	210	143	146	168	153	170
35	M	170	176	159	176	192	185
38	M	197	171	172	177	159	181
42	M	246	190	209	198	191	190
49	M	473	379	320	475	364	375
55	M	200	167	167	202	255	-
56	M	324	306	283	322	274	311

APPENDIX E_4

Serum triglyceride values - AHA Group

Subject No.	Sex	Months 0	1	2	4	6	12
08	F	174	99	101	91	98	94
10	F	163	105	188	109	174	-
29	F	154	137	256	161	166	144
37	F	340	218	317	262	264	294
47	F	218	159	243	209	185	-
50	F	91	-	115	60	52	101
53	F	158	225	167	188	-	334
04	M	118	97	94	114	80	87
05	M	312	193	197	236	129	190
09	M	139	180	165	124	167	111
11	M	168	199	199	203	144	293
13	M	217	183	172	155	123	130
14	M	125	121	85	121	108	162
19	M	193	229	150	163	176	146
23	M	102	67	81	89	85	78
32	M	176	119	146	185	108	130
34	M	117	84	63	61	114	85
35	M	170	114	107	152	164	108
38	M	100	98	93	77	96	96
42	M	162	218	178	142	165	139
49	M	>10,000	1530	1660	2920	725	800
55	M	196	144	162	224	234	-
56	M	375	297	317	502	415	578

APPENDIX E_4

Serum HDL values - AHA Group

Subject No.	Sex	Months 0	1	2	4	6	12
08	F	52	59	55	60	57	65
10	F	53	52	53	54	37	-
29	F	42	57	51	50	47	59
37	F	54	48	42	48	62	52
47	F	34	60	51	65	47	-
50	F	53	-	56	-	48	44
53	F	43	44	51	52	-	69
04	M	52	44	48	45	46	48
05	M	59	48	50	43	46	56
09	M	46	48	41	45	33	47
11	M	46	45	40	40	48	37
13	M	54	62	47	54	59	57
14	M	50	92	80	82	74	62
19	M	42	-	50	-	51	53
23	M	46	66	51	57	54	42
32	M	40	37	41	43	40	50
34	M	32	36	36	33	26	40
35	M	34	40	31	35	34	40
38	M	44	42	50	43	50	58
42	M	43	33	44	37	41	43
49	M	32	-	48	29	44	37
55	M	43	32	45	41	43	-
56	M	29	31	45	26	33	29

APPENDIX E_4

Serum LDL values - AHA Group

Subject No.	Sex	0	1	2	4	6	12
08	F	177	127	110	131	126	121
10	F	186	196	178	204	160	-
29	F	179	157	202	260	192	205
37	F	179	188	203	243	240	216
47	F	251	211	216	218	174	-
50	F	157	115	115	-	144	157
53	F	186	154	155	157	-	156
04	M	133	113	112	124	109	129
05	M	127	121	114	96	81	113
09	M	129	110	111	130	112	119
11	M	189	129	138	124	166	129
13	M	113	91	67	97	91	100
14	M	57	99	95	96	97	96
19	M	267	-	176	-	199	234
23	M	129	108	109	100	101	167
32	M	153	105	114	127	122	113
34	M	155	90	97	123	104	113
35	M	102	113	107	111	125	123
38	M	133	109	103	119	90	104
42	M	171	113	129	133	117	119
49	M	1559	-	60	138	175	178
55	M	118	106	90	116	165	-
56	M	220	216	175	196	158	166

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